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SEGMENTAL RESECTION FOR PULMONARY TUBERCULOSIS

An Analysis of 335 Cases

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SEGMENTAL RESECTION, made possible through the advent of antimicrobial therapy, is today an established and accepted procedure in the surgical treatment of pulmonary tuberculosis. The rate of sputum conversion has been found to be high and the morbidity and mortality rates are low. One of the greatest values of segmental resection is its preservation of pulmonary function.

Patients with a very limited pulmonary function have been accepted for segmental resection, e.g., resection of two segments and one subsegment on the only remaining left lung, following a previous pneumonectomy. Of 335 patients, 12 are dead and 170 have been restudied. Ninety-one patients were excluded, as the observation time was less than 1 year. Fifty-eight patients have refused re-investigation, mainly because they did not like to have another bronchospirometry. Of these, many are well and working full time but it has not been possible to obtain a personal restudy. Four patients from abroad are well and working but have not had postoperative lung-function tests. About half of the patients (47 per cent) had bilateral tuberculosis. The extent of the tuberculosis in the contralateral lung is shown in Table I. Seven patients had an open cavity in the contralateral lung. Sixty-one patients had had a cavity in the contralateral lung closed at the time of surgery: in 40 cases by a pneumothorax, in 17 by a thoracoplasty, and in 4 cases by conservative measures. In 17 per cent of the cases, it was necessary to do an extrapleural resection with a decortication of the remaining lung.

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TABLE I. THE CONDITION OF THE CONTRALATERAL LUNG AT THE TIME OF OPERATION

CONDITION OF THE CONTRALATERAL LUNG	NUMBER OF CASES
Normal	175
Old pleurisy	5
Small infiltration	67
Infiltration earlier treated by pneumothorax	15
Cavity "closed" by conservative treatment including chemotherapy	4
Cavity "closed" by pneumothorax	40
Cavity "closed" by thoracoplasty	17
Open cavity	4
Open cavity under pneumothorax or thoracoplasty	3
Segmental resection	3
Decortication and pneumonectomy	2
Total	335

Age is not in itself a contraindication for segmental resection in cavitary tuberculosis. The oldest patient, a 64-year-old man, tolerated well a segmental resection of the apical segment of the right upper lobe and a small thoracoplasty in one stage. Five years after operation he is in good condition and has negative guinea pig tests of the sputum and gastric washings. The reason for adding a thoracoplasty in this aged patient was emphysema and fibrosis of the remaining lung. The other 4 patients above the age of 60 also survived the operation.

The postoperative course must be carefully supervised in these older patients. Perfusion of blood through nonventilated areas of lung will decrease the arterial oxygen saturation, causing symptoms of coronary insufficiency. Whenever there is an insufficient ventilation in the postoperative course which cannot be immediately improved by more conservative measures, a tracheostomy with prolonged artificial ventilation should be instituted to guarantee the patient a normal oxygen and carbon dioxide tension.

Eight patients were operated on under the age of 20, the youngest being 9 years. In 6 of these young patients, no space-diminishing procedure was needed. In 1, an extrapleural pneumothorax space was developed at the end of the resection and filled with air to diminish the intrapleural space after the operation. In 1 patient, the diaphragm was mobilized around its periphery and resutured at a higher level. This latter operation is not recommended for general use as it results in impaired lung function. However, in the young and growing patients under the age of 20, it may occasionally be the best space-diminishing procedure. In the above case, there was 25 per cent of oxygen uptake on the left side after the procedure. The ventilation, however, was only 11 per cent. The age and sex distribution are shown in Table II and Fig. 1.

The indications for segmental resection are as follows: (1) tuberculous cavitation in 314 cases (94 per cent), (2) bronchiectasis with positive sputum in 5 cases, (3) bronchial stenosis in 2 cases, (4) tuberculous empyema with bronchial fistula in 2 cases, and (5) tuberculoma in 12 cases.

Preoperative Investigation of Pulmonary Function.—The most important information of pulmonary function is obtained by a study of all x-ray films from the beginning of the disease. The maximal movements of each diaphragm

TABLE II. AGE AND SEX DISTRIBUTION IN 335 PATIENTS UNDERGOING SEGMENTAL RESECTION FOR PULMONARY TUBERCULOSIS

AGE	MEN	WOMEN	TOTAL
0-9	1	—	1
10-19	3	4	7
20-29	33	42	75
30-39	69	46	115
40-49	70	29	99
50-59	28	5	33
60-69	3	2	5
Totals	207	128	335

is also recorded. When a segmental resection is intended, it is extremely important to examine frontal as well as lateral tomograms as pointed out by Chamberlain and Klopstock.[3] The maximal breathing capacity and, on certain indications, a bronchospirometry should be performed before the operation.

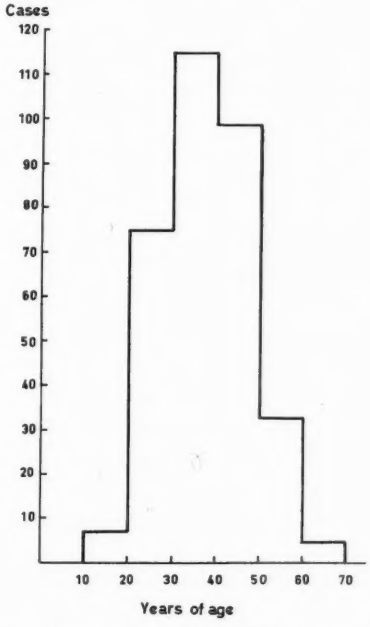

Fig. 1.—The age distribution in 335 cases of segmental resections for pulmonary tuberculosis.

Mortality.—Twelve patients died (Fig. 2). The operative mortality was 1.5 per cent. The cause of death in 3 patients was ventilatory insufficiency. One 59-year-old woman, with bilateral cavitary tuberculosis, died 2 days after operation in spite of artificial ventilation by respirator through a tracheostomy. One 41-year-old man had had a tuberculous cavity in the right lung treated by pneumothorax for 6 years. That treatment had resulted in a right-sided fibrothorax. There was a 10-year history of cavitary tuberculosis in the left lung treated for 5 years by pneumothorax. After the left-sided pneumothorax was abandoned,

there were two cavities, one in the upper lobe, and one in the superior segment of the lower lobe. In this patient, the apicoposterior segment of the left upper lobe, as well as the superior segment of the left lower lobe, were resected and at the same time an osteoplastic thoracoplasty was performed. Because of the thickened pleura over the right lung, the thoracoplasty and the extensive resection of the left lung, it was necessary to do a tracheostomy on the third day to add artificial respiration by respirator. This artificial respiration was needed for 8 days, after which the patient made an uneventful recovery. One month

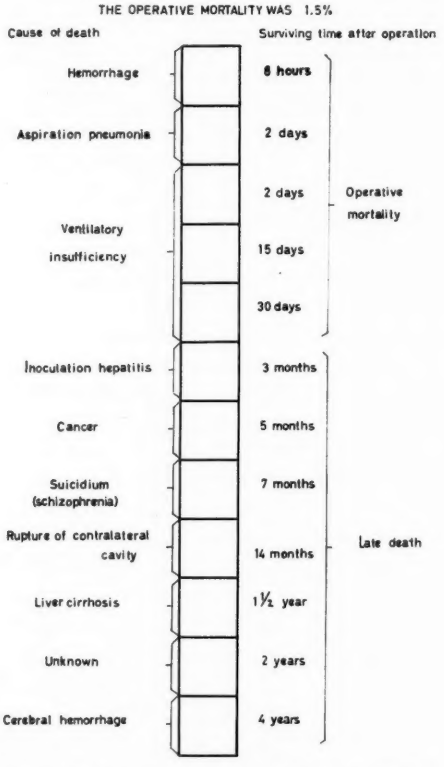

Fig. 2.—The operative mortality was 1.5 per cent (5 cases). There were 7 late deaths.

after operation a pulmonary edema developed, and he died suddenly. Autopsy showed lipoid degeneration of the myocardium, and right-heart insufficiency with failure and stasis. The third patient, who died from ventilatory insufficiency, was a 38-year-old man who had had a seven-rib thoracoplasty on the right side 6 years earlier. A resection of the apicoposterior segment of the left upper lobe plus a wedge of left lower lobe was performed for cavitary tuberculosis. On the fifteenth day after operation he went into coma and arterial puncture showed a carbon dioxide tension of 124 mm. Hg, and an oxygen tension of 42 mm. Hg. Tracheostomy was performed but it was too late. Before

operation, bronchospirometry showed a 72 per cent oxygen uptake on the left side. Cardiac catheterization, however, showed a pressure in the pulmonary artery of only 17 mm. Hg; after occlusion of the left pulmonary artery it rose to 32 mm. Hg mean pressure, and after occlusion of the right main pulmonary artery plus work, the mean pressure in the pulmonary artery rose further to 45 mm. Hg, with a systolic peak of 64. That patient had a maximal breathing capacity of 49 L. per minute preoperatively. Today such a patient would have been treated immediately after operation by tracheostomy and by artificial ventilation for at least 1 week. One patient died from hemorrhage, despite 13 units of blood by transfusion.

One 30-year-old patient, who 5 years earlier had had an extrapleural pneumothorax on the left side for cavitary tuberculosis, underwent resection of the posterior segment of the right upper lobe. After operation she vomited and died of an aspiration pneumonia.

Seven other patients in this series have died since operation. The causes of death included cirrhosis, carcinoma, hepatitis, suicide, cerebral hemorrhage, and 1 patient died following rupture of a tuberculous cavity on the right side 14 months after a segmental resection on the left. A 44-year-old man died 2 years after operation from an unknown cause.

Complications.—Nonfatal complications occurred in 15 per cent of cases of segmental resection.

1. *Air leakage* was the most important complication after segmental resection, and occurred in 10 per cent of the patients. Leakage occurred more frequently if either a decortication had been done for an old pneumothorax or if there had been an atypical resection, either wedge resection or subsegmental resection. Of these patients, 7.5 per cent were treated by closed drainage. In 2.5 per cent it was necessary to add further surgery, i.e., a thoracoplasty in 1 case, a thoracoplasty with decortication in another case, and in 2 cases it was necessary to do a resection with lobectomy plus chest wall extirpation. There were 2 cases of empyema. One healed by closed suction, in the other patient there was a downhill course with progression of disease in both lungs.

2. *Accidental opening of a tuberculous cavity* occurred in 3 cases without any late complication.

3. *Wound infection* was encountered in 3 cases but healed with chemotherapy.

4. *Ventilatory insufficiency* was encountered in 6 cases. In 2 patients a tracheostomy was performed; in 4 additional cases, it was necessary to add artificial ventilation by a respirator. This treatment was continued from 1 to 21 days.

5. *Trauma to the big vessels.* In 1 case, an incision was accidently made in the subclavian artery during the course of an osteoplastic thoracoplasty. The incision was closed by vascular suture. In 2 cases, the pulmonary artery was injured and in both cases repaired.

6. *Postoperative pain* radiating out in the arm was encountered in 1 patient who had had resection of the apicoposterior and anterior segments of the left upper lobe plus an osteoplastic roof-thoracoplasty, leaving the first rib in place. Thus, a scalenus anticus syndrome was produced and the patient was reoperated upon half a year later with resection of the first rib, after which the symptoms disappeared.

7. *Spillover with infiltrations in the contralateral lung* occurred in 1 case. Tracheostomy had to be performed and artificial ventilation with respirator was needed for 3 days, after which the patient made an uneventful recovery.

There were no postoperative complications in 85 per cent of the patients undergoing segmental resection for pulmonary tuberculosis.

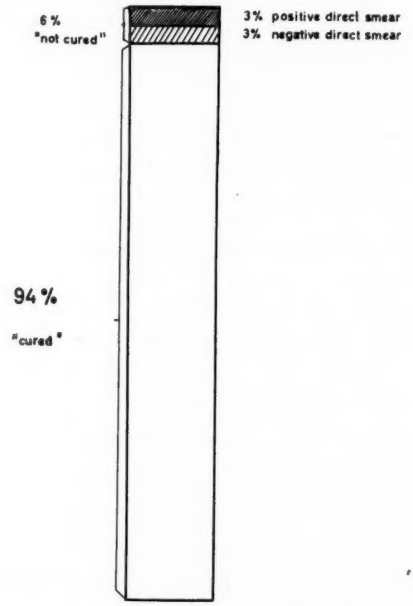

Fig. 3.—Ninety-four per cent were considered "cured." Only 3 per cent were positive on direct smear.

RESULTS

A patient was considered "cured" if (1) there was a negative guinea pig test on gastric washing and sputum, as well as negative cultures on sputum and a negative direct smear, on 3 consecutive days, and (2) no active disease was found at an x-ray examination. All these criteria were fulfilled in 160 patients of 170 patients restudied (94 per cent) (Fig. 3).

Of the 10 patients "not cured," 4 had a positive direct smear. This was due to reactivation on the contralateral side in 3 cases, and on the same side as the resection in 1 case. Five patients were negative on direct smear, but had

a positive guinea pig test. Four of these were working full time, and one performed a half day's work. Of the 5 patients who had positive guinea pig tests on gastric washings, 1 had a pneumothorax for cavitary disease on the contralateral side, 1 had a contralateral, extrapleural pneumothorax for cavitary disease, and a third patient had contralateral calcified pulmonary infiltration as well as bronchiectasis. The last patient in the group of those not cured was negative on direct smear and on guinea pig test of gastric washings, but had a positive culture of the sputum. She was working full time 6 years after operation, was free of symptoms, had a normal sedimentation rate, but x-ray studies showed some infiltration at the site of resection.

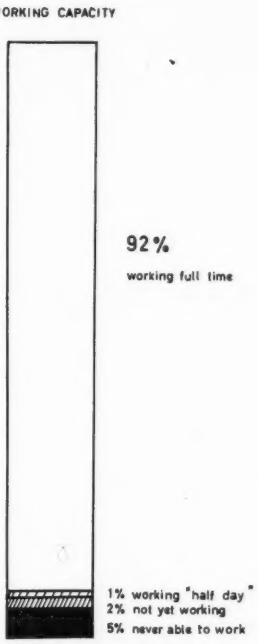

Fig. 4.—The working capacity.

Working Capacity (Fig. 4).—Of 158 "cured" patients, 146, or 92 per cent, were working full time 1 year after the operation. Many different professions were represented, of which housework and office work were most frequently encountered. Two patients performed a half-day's work and 10 did not work at all. Of these, 6 patients were considered unable to take up work again. Of these, 5 had had extensive bilateral tuberculosis with a maximal breathing capacity varying from 32 L. up to 70 L. per minute. One had a contralateral pneumothorax, 2 had contralateral 7-rib thoracoplasties, and 1 a 5-rib thoracoplasty. The remaining patient had gone through segmental resection without a thoracoplasty, but due to rheumatic arthritis in both shoulders she was unable to perform any work at all.

The arm movements were recorded and compared in 4 different groups of patients (Fig. 5). In a small group of 4 patients who had had a mobilization of the diaphragm as a space-diminishing procedure, there were normal arm movements in all. Pain following resection varied with the procedure (Fig. 6).

In the small group of 4 patients who had mobilization of the diaphragm as a space-diminishing procedure there were no complaints of pain.

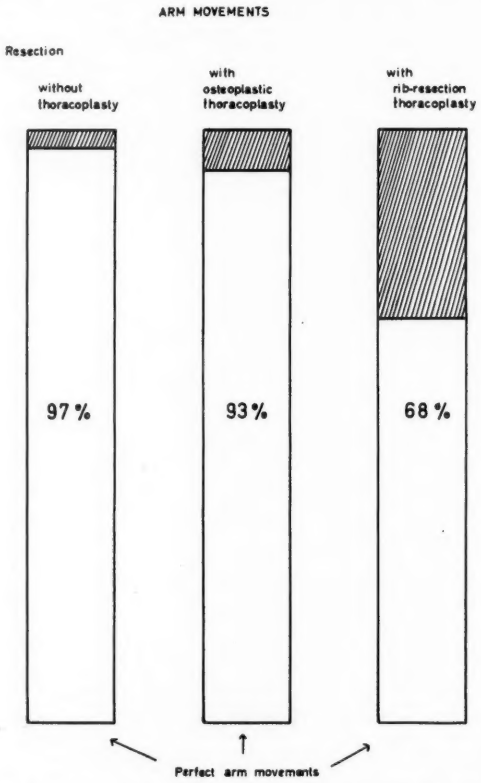

Fig. 5.—The best cosmetic and functional result regarding the arm movements was naturally found in patients undergoing resection without thoracoplasty. If a thoracoplasty was needed, the result was far better if an osteoplastic thoracoplasty was added than if a thoracoplasty by rib resection was performed.

Cough.—Seventy-four per cent of the patients did not have any cough at the time of restudy. The remaining 26 per cent experienced some irritating cough, usually called the smoker's cough, or they had some expectoration in the morning. In a few patients, suture granulomas were found around silk stitches, which had to be removed through the bronchoscope. In order to avoid this complication, it is the author's policy to use 30-day chromic catgut on atraumatic needles for bronchial closure and bronchotracheal anastomosis. This technique seems to be especially important in cases of pulmonary tuberculosis. A suture

granuloma may cause a positive guinea pig test which turns negative after removal of the suture. These suture granulomas have been proved to contain tuberculous tissue.

Dyspnea.—Eighty per cent of the patients did not feel any shortness of breath. The 20 per cent who complained of dyspnea were usually cases of far-advanced bilateral disease.

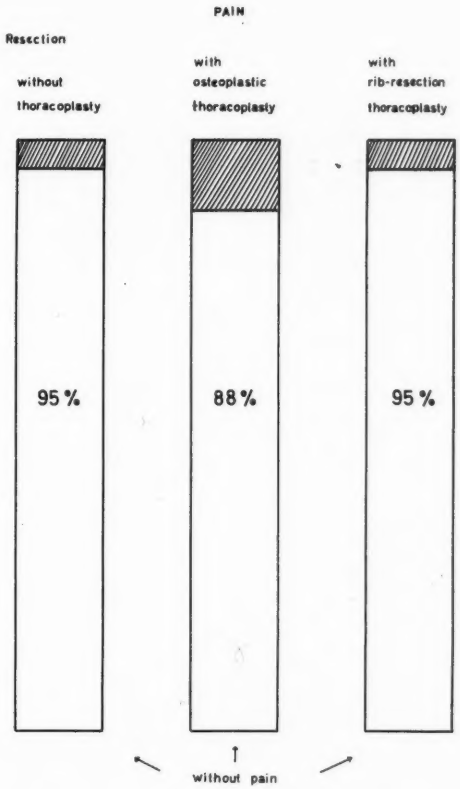

Fig. 6.—The incidence of pain was slightly higher in the patients having an osteoplastic thoracoplasty performed.

Functional Results (Figs. 8 and 9).—When the material is analyzed in order to find out the loss of function after the various segmental resections only unilateral cases have been included. It is obvious that parenchymal and pleural disease on the contralateral side will give a false bronchospirometric result. For instance, 1 patient showed a left-sided oxygen uptake of 60 per cent before and 53 per cent after resection of apicoposterior segments of the left upper lobe and an osteoplastic thoracoplasty due to pleural disease on the contralateral side.

Wedge resection can be performed without any significant loss of function. However, even the smallest resection may cause some impairment of function due to the fixation of diaphragm following most thoracotomies, in spite of breathing exercise. The more anatomic subsegmental resection is preferable to a wedge resection, in order to avoid dividing the drainage bronchus too close to the tuberculous lesion. A tuberculous endobronchitis in the divided little bronchus once caused infection and a fistula necessitating re-resection.

If one segment was removed the maximal breathing capacity decreased by 22 L. to 109 in male patients and by 16 L. to 77 in female patients.

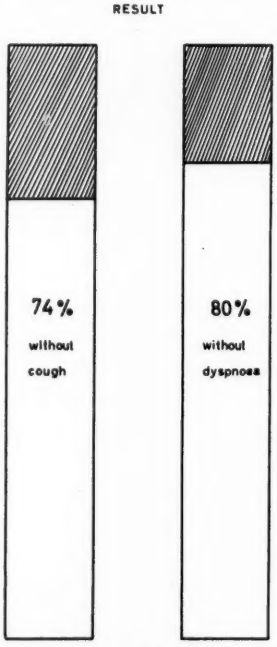

Fig. 7.—Twenty-six per cent of the patients had cough at the follow-up and 20 per cent were short of breath.

The maximal breathing capacity diminished 12 L. to 83 L. per minute in male patients undergoing resection of one segment with additional thoracoplasty. In those patients operated upon on the right side, the diaphragmatic movement was on an average 4.8 cm. as compared to 5.7 cm. on the left side.

Resection of one segment always causes some decrease of function on the operated side. When the resection is performed without thoracoplasty only a very slight decrease of oxygen uptake and ventilation is encountered. When a thoracoplasty is added there is a further decrease in oxygen uptake on the operated side although the ventilation remains approximately the same.

Resection of an additional subsegment to one segment did not cause any further reduction in pulmonary function.

Resection of 2 segments caused a decrease in the maximal breathing capacity of 26 L. to an average of 100 L. per minute in men (10 cases) and of 19 L. to 97 L. per minute in women (3 cases). If an osteoplastic thoracoplasty was added to the resection of 2 segments, there was a decrease of 23 L. to 94 L. per minute in men (5 cases). The addition of a thoracoplasty with rib resection in 3 men caused a decrease of 13 L. to 85 L. per minute.

The maximal diaphragmatic movements after resection of 2 segments on the right side was on an average 4.4 cm. on the right and 5.8 cm. on the left side (8 cases). After a left-sided resection, the maximal movements of the

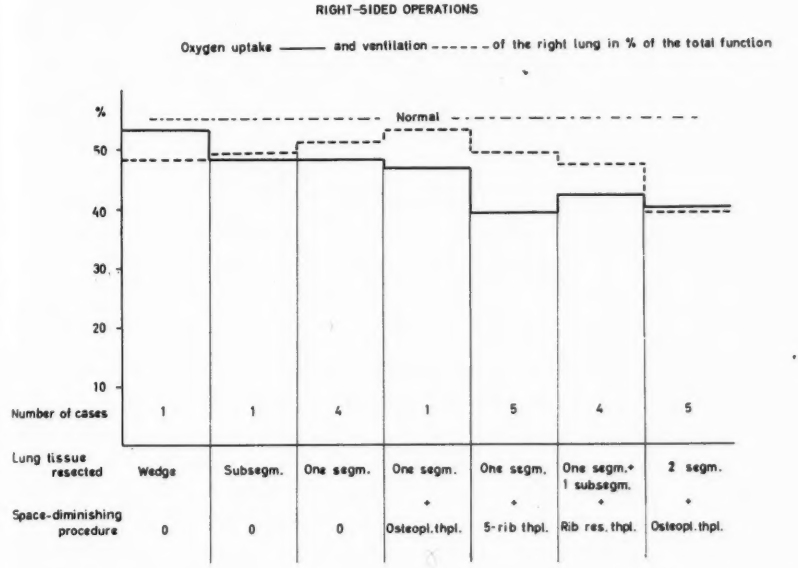

Fig. 8.—Diagram of the oxygen uptake and ventilation of the right lung in per cent of the total function after segmental resections on the right side. The segmental resection without thoracoplasty gives hardly any reduction in ventilation or oxygen uptake. Combined with thoracoplasty there is some reduction of oxygen uptake as well as of ventilation; however, the remaining lung function after these operations has been very good indeed.

diaphragm were 4.8 cm. on the right as compared to 4.0 cm. on the left side (4 cases). If an osteoplastic thoracoplasty was added to a right-sided resection of 2 segments, the diaphragmatic excursion was measured to an average of 5.9 cm. on both right and left sides in 7 cases. In 6 cases of a similar left-sided procedure, the right diaphragm moved 5.5 and the left 5.0 cm. (6 cases). Addition of a thoracoplasty with rib resection to resection of 2 segments on the left side resulted in a movement of 5 cm. on the right and 3.5 cm. on the left side in 2 cases.

The oxygen uptake and ventilation are only slightly decreased after resection of 2 segments. If a thoracoplasty was added, either an osteoplastic type or by rib resection, a significant decrease in both oxygen uptake and ventilation was observed.

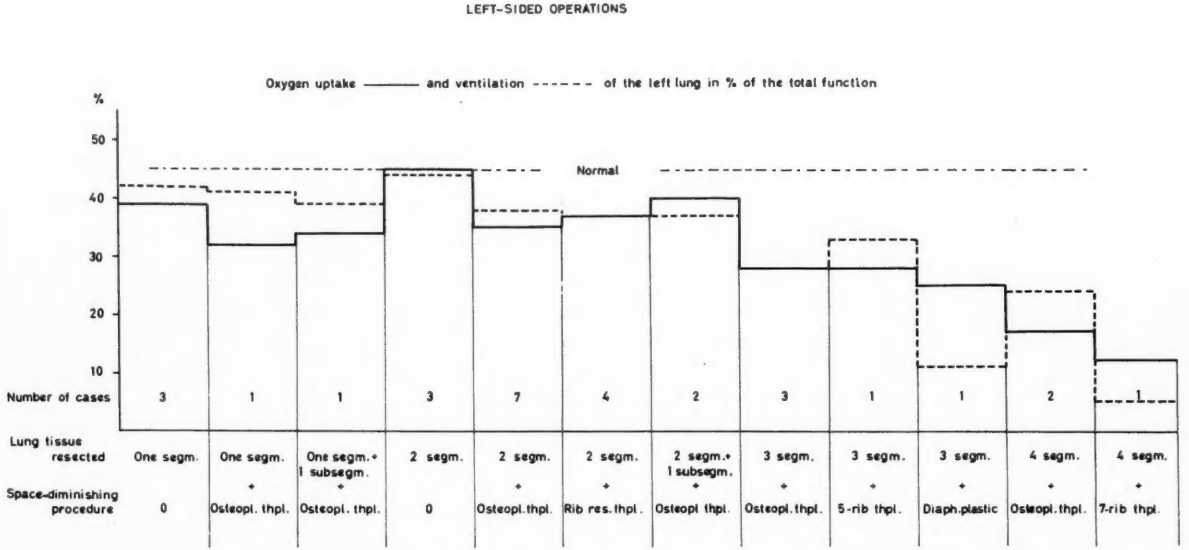

Fig. 9.—Diagram of the oxygen uptake and ventilation of the left lung in per cent of the total function after segmental resections on the left side. For resection of up to 2 segments there is only an insignificant reduction of function of the operated side. If more lung tissue is resected the impairment of lung function is correspondingly increased.

Resection of 3 segments and a thoracoplasty caused a decrease in the maximal breathing capacity of 42 L. to 91 L. per minute in 5 cases.

After resection of 3 segments with a space-diminishing procedure, there was a significant decrease of oxygen uptake and ventilation on the operated side. Different space-diminishing procedures did not influence the oxygen uptake, but the ventilation was most decreased in the patient having a mobilization of the diaphragm.

Resection of 4 segments with an osteoplastic thoracoplasty caused a marked drop in oxygen uptake and in ventilation on the operated side (Figs. 8 and 9).

SUMMARY

An analysis is made of 335 cases of segmental resection for pulmonary tuberculosis. The operative mortality (including patients with marked ventilatory insufficiency) was 1.5 per cent. Of the 170 patients followed from 1 to 6 years, it has been gratifying to find 97 per cent with a negative direct smear and 94 per cent with a negative culture and guinea pig test of sputum and gastric washings. Ninety-two per cent were working full time. The incidence of impaired arm movements, pain, cough, and dyspnea is recorded. The best cosmetic result and best arm movements were obtained when no thoracoplasty was added. The diaphragm mobilization gave the best cosmetic but the worst functional result of the different space-diminishing procedures. The osteoplastic thoracoplasty gave a better cosmetic result and better arm movements when compared with a thoracoplasty with rib resection. There was a higher incidence of postoperative pain in the group in which rib resection thoracoplasty was performed. The late functional result has been very encouraging and it has been proved that segmental resections can be performed with a minimal loss of function. The contralateral side has withstood the resection very well.

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AN ANALYSIS OF THE PULMONARY FUNCTION OF NINETY PATIENTS FOLLOWING PNEUMONECTOMY FOR PULMONARY TUBERCULOSIS

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SINCE it has become evident that special cases of pulmonary tuberculosis can be treated adequately by pneumonectomy, several authors have published data concerning the influence of this operation on pulmonary function.[2, 4, 5, 7-11, 13-15]

From the functional point of view, the consideration as to whether a pneumonectomy is justifiable or not depends on both the results of the preoperative function tests and of the expected changes of the function of the remaining lung in the early and late postoperative period. An analysis of the results of these tests in patients who underwent a pneumonectomy several years ago is, therefore, of special importance in determining which patients can be treated in the future by pneumonectomy without risks with respect to pulmonary function.

From various publications, it is clear that, in most patients, distention of the lung tissue in the postoperative period occurs. It could not be proved, however, that distention resulted in pulmonary emphysema.[5, 7, 8, 9, 14] With regard to the interpretation of distention after pneumonectomy, opinions differ. Most authors consider distention as an unfavorable condition with respect to the pulmonary function.[2, 5, 15, 16] Cournand[5] and Birath[2] and their associates regarded pulmonary distention as a condition in which the ventilatory capacity is limited and the respiratory efficiency is reduced. Rienhoff,[14] on the other hand, interpreted distention as a physiologic phenomenon and regarded any method employed to impede this dilatation as unprofitable.

The performance of a thoracoplasty, with the intention of facilitating the adaptation of the remaining lung to the modified intrathoracic conditions, is considered undesirable by most authors.[7, 8, 9, 14] Rossier and Bühlmann[15, 16] on the contrary, hold the view that the best results are obtained by preventing the distention as much as possible. They state that the combination of pneumonectomy and thoracoplasty ("Deckplastik") always leads to a striking increase of the ventilatory economy, even in patients with a pre-existing thoracoplasty.

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The purpose of this paper is to discuss the results of the pulmonary function tests in a group of 90 tuberculous patients at an average period of 52 months after pneumonectomy and to compare these results with those published by other authors.

MATERIAL

The patients examined belonged to a group of 125 patients in whom Eerland[10] and co-workers performed a pneumonectomy for pulmonary tuberculosis in the years between 1947 and 1954. At the time of the follow-up study, 105 patients were alive. As of December, 1956, 90 patients submitted voluntarily to a follow-up study of their pulmonary function. The function of 4 additional patients was studied after this date. Eleven patients refused. Of the 90 patients, the functional data just before the pneumonectomy, at the time of the discharge from the hospital and those at the time of the follow-up study, were available. The time interval between operation and discharge ranged from 5 to 30 months (mean, 11 months), whereas the time between operation and follow-up study ranged from 29 to 84 months (mean, 52 months).

In Table I, the age groups for each of the sexes at the time of the operation and at the follow-up are given. The average age at the time of the pneumonectomy was 30 years and 8 months, at the follow-up 35 years.

TABLE I. AGE OF PATIENTS BEFORE PNEUMONECTOMY (BP) AND AT THE TIME OF FOLLOW-UP EXAMINATION (FU)

AGE GROUPS IN YEARS	FEMALE		MALE		TOTAL	
	BP	FU	BP	FU	BP	FU
15-19	6	1	2	—	8	1
20-29	15	11	19	7	34	18
30-39	21	24	20	24	41	48
40-49	2	7	4	10	6	17
50-59	—	1	1	5	1	6
Total	44	44	46	46	90	90

The final period of treatment previous to the operation averaged 59 months and the mean interval between the discovery of the tuberculosis and the pneumonectomy was 103 months. In most of the cases, an illness of long duration preceded the operation and, in spite of prolonged bed rest and many forms of drug therapy or active treatment, slow progression took place, not uncommonly leading to total destruction of the tissue of one of the lungs.

In Table II, the roentgenologic status of both the resected and the contralateral lung at the time of pneumonectomy is given. At the time of the follow-up, the clinical status of 87 patients was good while, in 3 cases, the tuberculous lesions were not yet fully cured.

The analysis of the function data of the 90 patients is hampered by the multitude of factors which can be active. We have explored 5 of these: (1) sex, (2) whether remaining lung is right or left, (3) the presence or

TABLE II. THE ROENTGENOLOGIC LESIONS IN THE LUNGS AT THE TIME OF THE PNEUMONECTOMY

RESECTED LUNG	CONTRALATERAL LUNG				TOTAL
	NO LESIONS	INDURATIVE LESIONS			
		MINIMAL	MODERATE	EXTENSIVE	
Destroyed	11	16	7	1	35
Empyema	6	4	4	3	17
Extensive lesions	9	10	4	—	23
Moderate lesions	4	6	3	—	13
Minimal lesions	2	—	—	—	2
Total	32	36	18	4	90

absence of thoracoplasty, (4) asthmatic symptoms, and (5) diaphragmatic paralysis (Table III).

TABLE III. DISTRIBUTION OF THE 90 PATIENTS IN FIVE PAIRS OF FACTORS INFLUENCING THE PULMONARY FUNCTION

PAIRS OF FACTORS		♂	♀	Rr	Lr	Ta	Tp	Aa	Ap	Pa	Pp
Male patients	♂	*46*	—	30	16	19	27	30	16	39	7
Female patients	♀	—	*44*	31	13	33	11	27	17	34	10
Remaining right lung	Rr	30	31	*61*	—	35	26	43	18	52	9
Remaining left lung	Lr	16	13	—	*29*	17	12	14	15	21	8
Thoracoplasty absent	Tp	19	33	35	17	*52*	—	33	19	35	17
Thoracoplasty present	Ta	27	11	26	12	—	*38*	24	14	38	0
Asthmatic factors absent	Aa	30	27	43	14	33	24	*57*	—	47	10
Asthmatic factors present	Ap	16	17	18	15	19	14	—	*33*	26	7
Diaphragmatic paralysis absent	Pa	39	34	52	21	35	38	47	26	*73*	—
Diaphragmatic paralysis present	Pp	7	10	9	8	17	0	10	7	—	*17*

Thoracoplasty was not performed in 52 patients (Group I) and was done in 38 (Group II). In 27 patients of Group II, the thoracoplasty was performed before the pneumonectomy. In most of these patients, thoracoplasty was done because resection therapy was not yet available. Very active forms of tuberculosis and empyema—both frequently combined with drug resistance—formed the remaining indications. Thoracoplasty before or during the pneumonectomy was only done for empyema. The extent of the thoracoplasty varied from 4 to 10 ribs, as follows:

Number of ribs partly resected	4	5	6	7	8	9	10
Number of patients	12	2	3	7	7	1	6

In Figs. 1-8, asthmatic patients are pointed out by the use of a segment of a circle below the circle itself. The distribution of asthmatic patients in Groups I and II is almost equal (Table III).

METHODS

For the spirometric recordings, the spirometer designed by Dirken and Orie* was used.[17] All measurements were performed with the patients in the supine position. The volumes are given at ATPS.†

*Manufactured by Lode N. V., Groningen, The Netherlands.
†Ambient temperature and pressure saturated with water vapor.

The vital capacity was determined by measuring the maximum volume inspired after a complete expiration. The VC* was measured in triplicate and the highest value was regarded to be the best one.

The timed vital capacity in one second was measured according to the technique of Tiffeneau.[18] The TVC was determined at least in quadruplicate and the highest value obtained was taken for the calculations. The TVC is not only expressed as a percentage of the VC—TVC/VC ratio—but also as a percentage of the TLC—TVC/TLC ratio—for a reason explained later.

The total lung capacity was obtained by adding the residual volume to the vital capacity.

The maximum breathing capacity was measured at 30 breaths per minute. This frequency was indicated by the examiner. In the case of a slight difference in that frequency, the values were corrected. This technique has the following advantages: (a) 30 breaths per minute is for most patients the optimum breathing frequency during exercise; (b) the data are reproducible and have a better mutual comparability; (c) the FRC during hyperventilation (MBC) is accessible to mutual comparison; and the MBC can be compared with the predicted MBC, which can be calculated from the TVC by multiplying it by 30 ($MBC_{predicted} = 30 \times TVC$).

Before the operation and at the time of discharge of the patients, the FRC was measured by a closed system method. At the resting respiratory level, the patients were connected with a spirometer system of known volume, filled with nearly pure oxygen. By mixing this oxygen with the gas in the lungs, the oxygen-concentration in the system decreased and the extent of the fall is a measure for the volume of the gas in the lungs at the moment of the connection to the spirometer system. During the whole determination the volume of the lung-spirometer system was kept nearly constant by compensating the oxygen uptake of the patient. The gas analysis was performed by measuring continuously the change of the thermal conductivity of the gases

*The following abbreviations are used in this paper:

TLC	= total lung capacity in milliliters.
VC	= vital capacity in milliliters.
TVC	= timed vital capacity or volume expired during the forced vital capacity maneuver, in the first second in milliliters.
FRC	= functional residual capacity in milliliters.
FRC^r	= FRC in rest.
FRC^h	= FRC during hyperventilation.
RV	= residual volume in milliliters.
ERV	= expiratory reserve volume in milliliters.
MBC	= maximum breathing capacity in liters.
RV/TLC	= ratio of RV to the TLC in per cent.
FRC/TLC	= ratio of FRC to the TLC in per cent.
TVC/TLC	= ratio of TVC to the TLC in per cent.
TVC/VC	= ratio of TVC to the VC in per cent.
bp	= value before the pneumonectomy.
ad	= value at the discharge from the sanatorium.
fu	= value at the time of the follow-up study.
VC_{br}	= the VC of the contralateral lung before the operation as calculated from the VC_{bp} and its bronchospirometrically found percentage.

in the spirometer by means of a diaferometer.*[20] At the follow-up study the FRC was measured by an improved closed circuit method using helium—2.8 per cent in room air—as an indicator gas.[12, 17, 21] A detailed description of this method is given elsewhere.[17] In only 12 patients was the FRC at the follow-up examination determined by the original technique.† Since, in the original technique, no correction was made for the nitrogen elimination from the blood, the values obtained by the modified technique were somewhat smaller—about 10 per cent.[17] The values of the FRC are the mean values of determinations in duplicate, in such a way that only differences less than 200 ml. were accepted.

The FRC during hyperventilation—FRC^h—was obtained by adding the change of the expiratory level, which may occur during the performance of the MBC, to the FRC in rest—FRC^r. A rise of the expiratory level results in an increase of the FRC, and a shift of the level to the expiratory side results in a decrease in the FRC.

The residual volume was obtained by subtracting the expiratory reserve volume from the FRC^r ($RV = FRC^r - ERV$).

Bronchospirometry was performed preoperatively in 61 patients. In most cases, the Carlens catheter and the McKesson double spirometer, filled with oxygen, was used. Since 1954, the Lode double spirometer is used. Before 1950, bronchospirometry was performed with the Zavod catheter.

The normal values for the VC are calculated according to Anthony from the tables of Harris and Benedict for basal metabolism.[1] In the statistical treatment of the material, the various standard errors (S.E.) and the correlation coefficient (r) are calculated with the methods indicated by Bradford Hill.[3] If two means or two proportions differed by more than twice the value of the standard error of the difference, the difference is said to be "significant." In the instances when it was three times its standard error, it was said to be "highly significant." $p = 0.05$ is taken as level of significance; S_r = the standard error of the correlation coefficient.

RESULTS

The Vital Capacity.—

1. An impression of the changes in the VC as a result of the removal of the affected lung is obtained by comparing the VC_{bp} (before pneumonectomy) with the VC_{fu} (follow-up). In Fig. 1 the VC_{bp} is plotted against the VC_{fu} for each patient. The points falling at the right of the line $VC_{bp} = VC_{fu}$ indicate patients with a greater VC at the follow-up than before the operation, those falling at the left indicate the reverse situation. In 87 per cent of the cases, the VC_{fu} was smaller than the VC_{bp}.

2. In the same way, as in Fig. 1, a comparison is made between the VC_{fu} and the VC_{br} (Fig. 2). In this graph, the data of only 61 patients are given,

*Manufactured by Kipp N. V., Delft, The Netherlands.

†In 12 patients, the function tests were performed in the Department of pulmonary diseases of the State University at Groningen. Chief: Prof. N. G. M. Orie, M.D.

owing to the fact that bronchospirometry was not performed before the operation in every case. In 67 per cent of the patients without a thoracoplasty (Group I), the VC_{fu} exceeded the VC_{br}; while this occurred in only 29 per cent of the cases with a thoracoplasty (Group II). This difference between the two groups proved to be statistically highly significant (S.E. 0.131, difference 0.394). The mean values of the VC_{fu} and the VC_{br} of these 61 patients

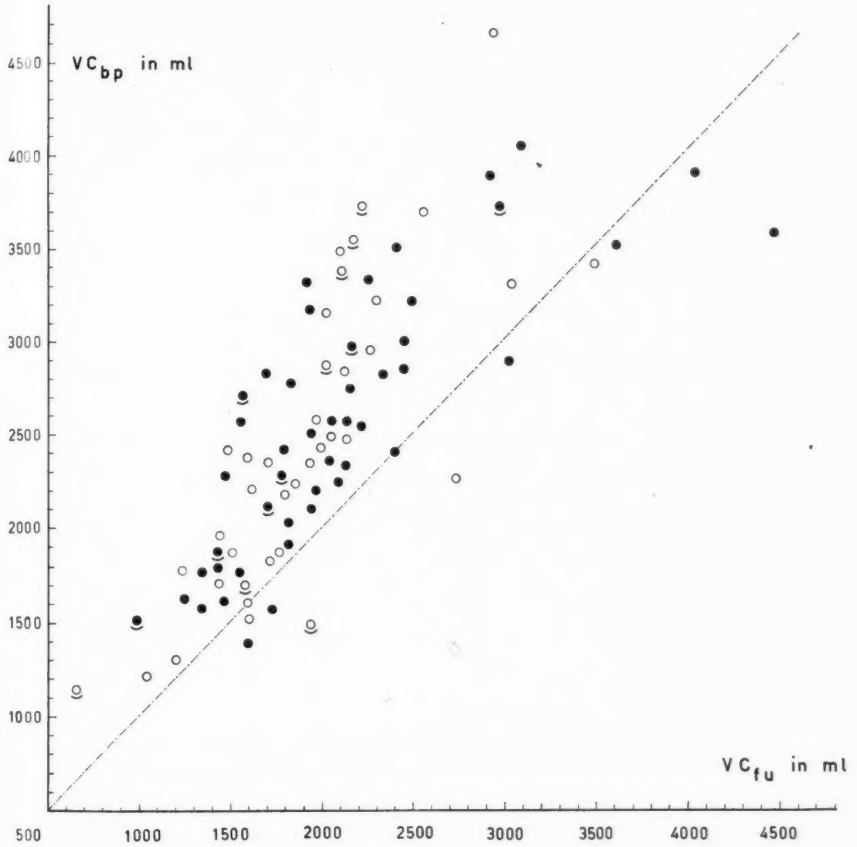

Fig. 1.—Comparison of the VC before the pneumonectomy (VC_{bp}) and at the follow-up (VC_{fu}).
● Patients without a thoracoplasty (Group I).
○ Patients with a thoracoplasty (Group II).
∪ Patients with asthmatic symptoms.

are given in Table IV. These data suggest that the decrease of the VC of the contralateral lung in Group II is due mainly to the values of the patients who underwent a thoracoplasty after the pneumonectomy.

3. In Table V, the 61 patients of Table IV are divided into three equal groups, according to the extent in which the resected lung took part in the

TABLE IV. THE MEAN VALUES OF THE VITAL CAPACITY OF THE REMAINING LUNG IN MILLILITERS

	GROUP I	GROUP II			TOTAL
			THORACOPLASTY BEFORE AND AFTER PNEUMONECTOMY		
	40 PERSONS	21 PERSONS	15 PERSONS (BEFORE)	6 PERSONS (AFTER)	61 PERSONS
Calculated according to Anthony[1]	1,740	1,900	1,880	1,940	1,800
VC_{br}	1,970	2,060	1,990	2,210	2,000
VC_{fu}	2,130	1,970	1,970	1,970	2,070

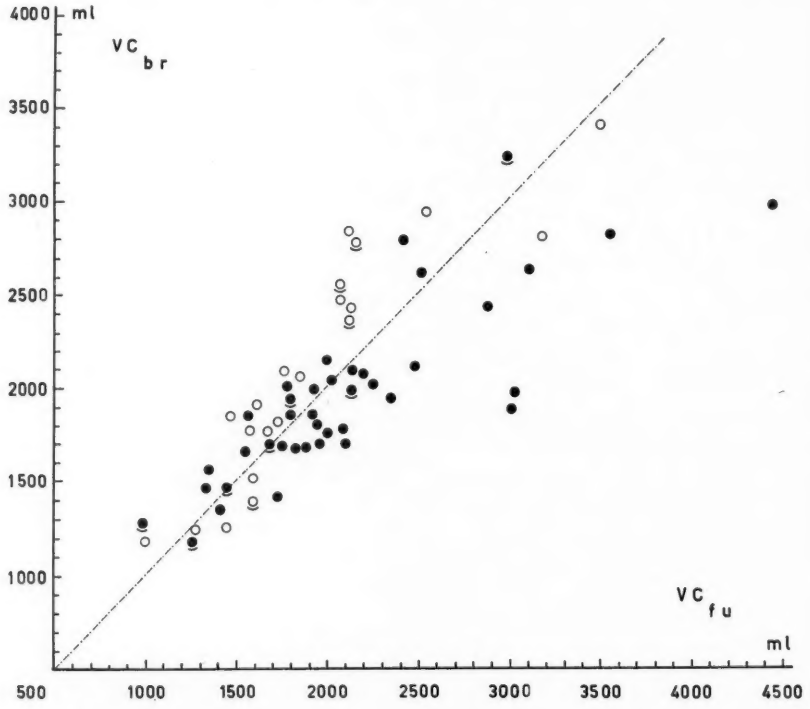

Fig. 2.—Comparison of the VC of the remaining lung before the pneumonectomy (VC_{br}) and at the follow-up (VC_{fu}).

VC_{bp}. By comparing the percentages of expected and actual loss of the VC in these groups, the following conclusions can be drawn: (a) In the group with a mean expected loss of 6 per cent, the actual loss was significantly higher ($p = 0.001$). In this group of 21 patients, 11 had a thoracoplasty before or during the pneumonectomy and, in 3 patients, a thoracoplasty was performed after the pneumonectomy. (b) In the group in which the participation of the resected lung ranged between 15 and 26 per cent, the expected loss corresponds with the actual loss. (c) In the group with the mean expected loss of 34 per cent, the actual loss is significantly smaller ($p = 0.01$). In the latter two

TABLE V. THE RELATION BETWEEN THE MEAN EXPECTED LOSS ($VC_{bp} - VC_{br}$) AND THE MEAN ACTUAL LOSS ($VC_{bp} - VC_{fu}$)

NUMBER OF CASES	PARTICIPATION OF THE RESECTED LUNG IN THE VC_{bp}	MEAN EXPECTED LOSS AS PER CENT OF THE VC_{bp}	MEAN ACTUAL LOSS AS PER CENT OF THE VC_{bp}
21	0-14%	6%	16%
20	15-26%	20%	19%
20	27-45%	34%	26%

groups (b and c), there were only three and four thoracoplasties, respectively; 5 before or during the pneumonectomy and only one after the resection.

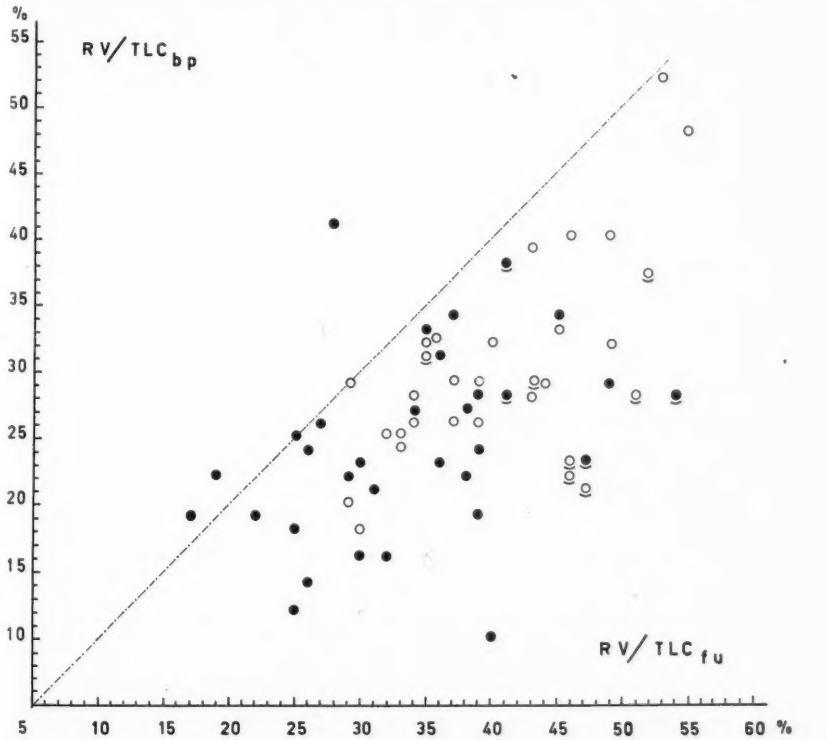

Fig. 3.—Comparison of the RV as a percentage of the TLC before the pneumonectomy (RV/TLC_{bp}) and at the follow-up (RV/TLC_{fu}).

The Residual Volume.—

An alteration of the RV/TLC ratio can be due to a change in the RV, a change of the VC, or an alteration of both volumes mentioned, because

$$RV/TLC = \frac{RV}{RV + VC}.$$

In order to get an impression of the changes occurring after pneumonectomy, the RV/TLC_{fu} ratio for each patient is compared with the RV/TLC_{bp} and the RV/TLC_{ad} ratio; the RV_{fu} with the RV_{bp}.

1. In Fig. 3, the RV/TLC_{bp} ratio is plotted against the RV/TLC_{fu} ratio for 63 patients; in 27 patients no RV/TLC_{bp} ratio was available. From the distribution of the points, the following conclusions can be derived: (a) in 92 per cent of the cases, the RV/TLC_{fu} ratio is greater than the RV/TLC_{bp} ratio, and (b) before the pneumonectomy, the difference between the mean RV/TLC ratios of both groups is statistically significant ($p = 0.01$). The same

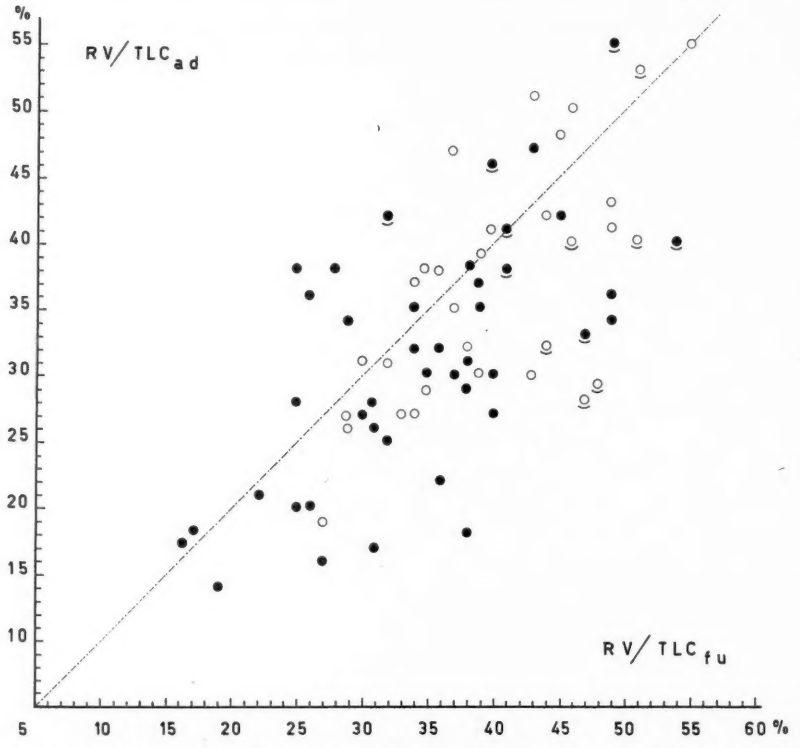

Fig. 4.—Comparison of the RV as a percentage of the TLC at the time of discharge from the sanatorium (RV/TLC_{ad}) and at the follow-up (RV/TLC_{fu}).

holds true for the mean ratios at the follow-up ($p = 0.04$). The change of the mean RV/TLC ratio caused by the resection is in both groups of the same proportion and proved to be statistically significant (Group I, $p = 0.01$; Group II, $p = 0.03$). The mean values are given in Table VII.

2. The change of the RV/TLC ratio in the period between the discharge from the hospital and the follow-up examination is given in Fig. 4. The distribution of the points in this figure shows an increase of the RV/TLC ratio in 65 per cent of the cases, equally distributed over the two groups. From this it is concluded that the difference between the RV/TLC ratios for Groups I and II did not change significantly following discharge from the hospital ($p = 0.3$).

3. Since the increase of the RV/TLC ratio can be due both to an increase of the RV and to a decrease of the VC, it is necessary to study the changes of the absolute RV values. In Fig. 5, the changes of the absolute values of the RV, before and after pneumonectomy, are given. In 70 per cent of the cases, equally distributed over the two groups, an increase of the RV was found. Consequently, the increase of the RV/TLC ratio after pneumonectomy was, for the greater part of the cases, not only due to a decrease of the vital capacity (Fig. 1) but also to an increase of the residual volume.

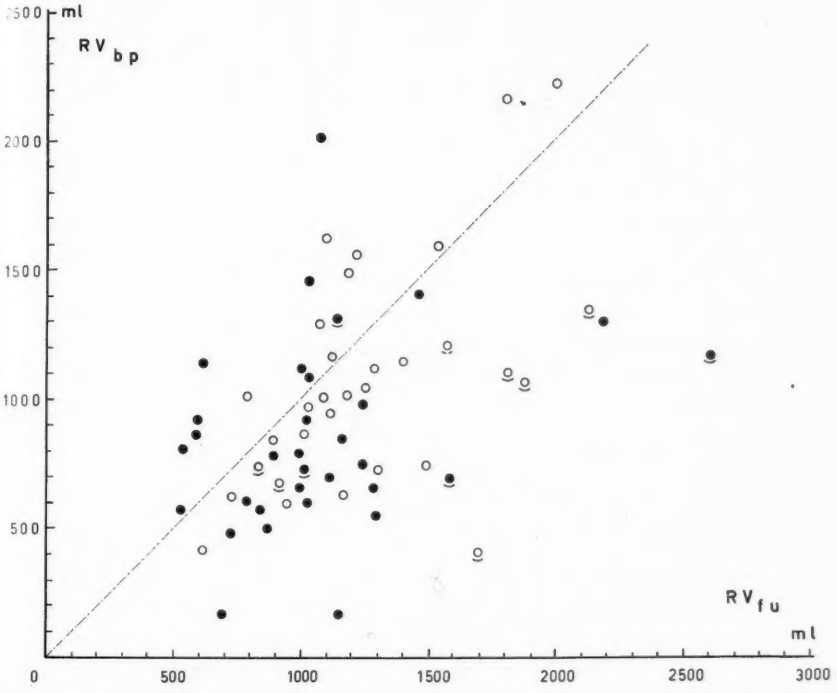

Fig. 5.—Comparison of the RV in milliliters before the pneumonectomy (RV_{bp}) and at the follow-up (RV_{fu}).

4. The increasing age of the patients must be taken into account in judging the change of the RV/TLC ratio in the postoperative period. The average interval between the discharge from the sanatorium and the follow-up examination amounts to 41 months. Within this period, the mean RV/TLC ratio of Group I changed from 31 to 34 per cent and that of Group II from 37 to 40 per cent. It is improbable that these differences can be explained by the slight increase in age.

Reviewing these data, the conclusion can be drawn that in both groups the increase of the RV/TLC ratio during the first 11 months after the pneumonectomy (Period bp/ad) is mainly due to a decrease of the VC, while the RV is relatively constant. In the period of about 41 months after the discharge (Period

ad/fu), the increase of the RV/TLC ratio is due to an increase of the RV, while the VC is almost constant. This phenomenon is graphically demonstrated in Fig. 6.

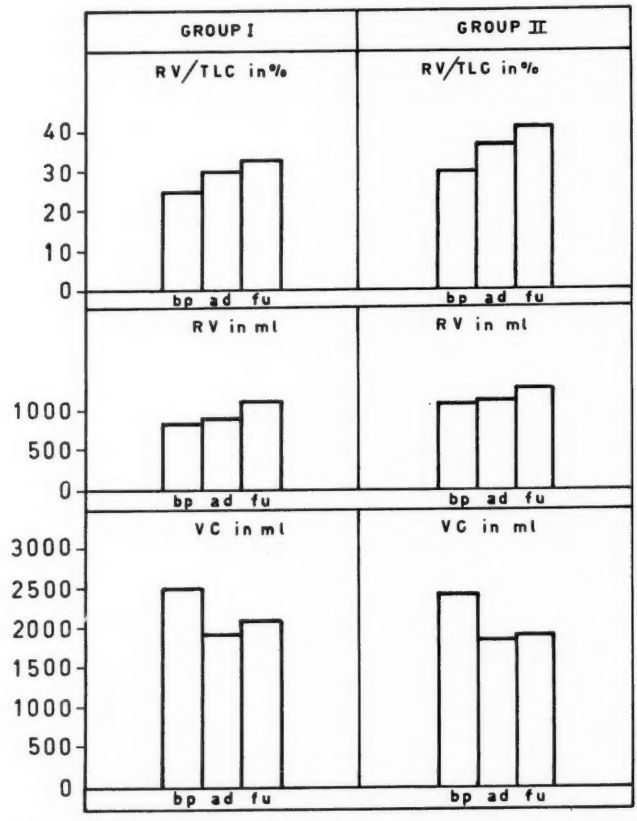

Fig. 6.—Comparison of the VC in milliliters, RV in milliliter and the RV/TLC ratio, before pneumonectomy (bp), at discharge (ad), and at follow-up (fu).

The Functional Residual Capacity.—

The FRC at rest and during hyperventilation, before the pneumonectomy and at the follow-up examination, is related to the ERV (FRC – RV) and to the shift of the expiratory level during maximal hyperventilation (Table VI). Some remarkable differences between the patients without (Group I) and with a thoracoplasty (Group II) were found: (a) In Group I the RV increased significantly ($p = 0.04$) in the period bp/fu, while the ERV was constant. In Group II, on the contrary, the significant increase of the RV ($p = 0.04$) was coupled with a comparable significant decrease of the ERV ($p = 0.02$). (b) During hyperventilation, MBC, hardly a rise of the

expiratory level occurred in Group I, while before the pneumonectomy this shift was highly significant ($p = 0.005$). In Group II, the shift of the expiratory level was evident both before and after the pneumonectomy.

TABLE VI. THE SUBDIVISIONS OF THE FRC AT REST (FRC^r) AND DURING HYPERVENTILATION (FRC^h) IN MILLILITERS

	POINT OF TIME	RV	ERV	FRC^r	FRC^h	SHIFT OF THE EXPIRATORY LEVEL
Group I	bp	860	490	1,350	1,660	+ 310
	fu	1,050	510	1,560	1,620	+ 60
Group II	bp	1,070	530	1,600	1,980	+ 380
	fu	1,280	340	1,620	1,960	+ 340
Total	bp	950	520	1,470	1,840	+ 370
	fu	1,110	390	1,500	1,680	+ 180

The Timed Vital Capacity.—

Generally, the TVC is expressed in relation to the VC—TVC/VC ratio. In our material it became evident that, in both groups of patients, the ratios before the pneumonectomy and those at the follow-up were equal (Table VII)—the TVC decreased in the same measure as the VC. Moreover, it could be proved from the TVC curves that the maximum volume of gas, expired in every space of time within one second, was diminished in proportion to the alteration of the VC. The question arises, however, as to whether or not the constancy of the TVC/VC ratio means that the pulmonary function with regard to the TVC changed only quantitatively. When performing the TVC, the expiratory forces do not act upon a gas volume that equals the VC, but upon a volume equivalent with the TLC. Therefore, it seemed more reasonable to express the TVC in relation to the TLC (TVC/TLC ratio). By calculating the TVC/TLC ratio before the pneumonectomy and at the follow-up, a statistically significant difference between both values (Group I, $p = 0.04$; Group II, $p = 0.05$) could be shown (Table VII). The differences between the TVC/TLC ratios of both groups before the operation as well as at the follow-up study proved to be significant ($p_{bp} = 0.004$; $p_{fu} = 0.005$).

TABLE VII. THE MEAN VALUES OF THE RV/TLC, TVC/VC, AND THE TVC/TLC RATIOS AND THE MBC IN PATIENTS WITH AND WITHOUT A THORACOPLASTY IN ADDITION TO A PNEUMONECTOMY

	POINT OF TIME	RV/TLC	TVC/VC	TVC/TLC	MBC IN L./MIN.	
					ACTUAL	PREDICTED
Group I	bp	25	66	51	49	51
	fu	34	66	44	39	41
Group II	bp	30	60	42	44	44
	fu	40	61	37	34	36

The Maximum Breathing Capacity.—

In both Groups I and II, there was a significant decrease of the MBC in the period between the pneumonectomy and the follow-up (Group I, $p = 0.005$;

Group II, p = 0.003. The relation between the actual and the predicted value of the MBC was a very close one (r = 0.978; Sr = 0.100) (Table VII).

The Relation Between the TVC and the RV.—

In order to get an impression about the relation between the TVC/TLC and the RV/TLC ratios in the patients at the time of the follow-up, the correlation diagram for these data is constructed (Fig. 7). There is a definite negative correlation between the RV/TLC and the TVC/TLC ratio (r = –0.816;

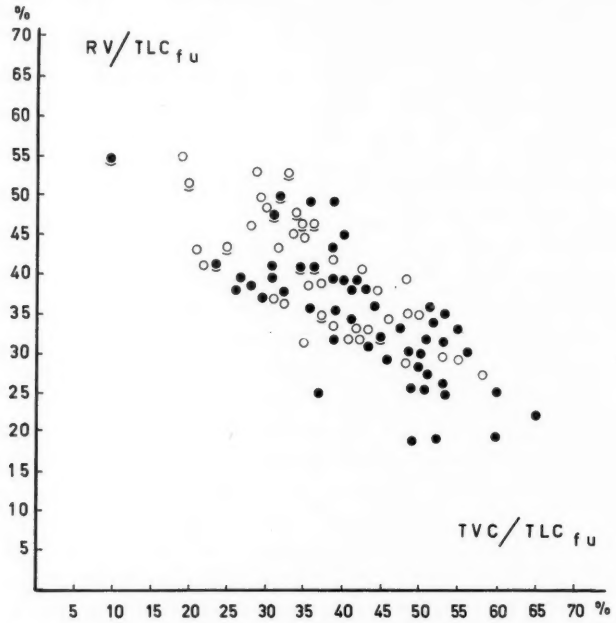

Fig. 7.—Relation between the RV/TLC ratio and the TVC/TLC ratio at the follow-up examination.

Sr = 0.036) and this correlation proved to be far better than that between the formerly used TVC/VC and the RV/TLC ratio (r = –0.456; Sr = 0.084) (Fig. 8). Since in Fig. 7 both the RV and the TVC are related to the TLC, a good correlation between the absolute values of the RV and TVC is present too. The amount of gas that maximally can be expired in the first second after a maximal inspiration is evidently correlated to the residual volume in patients without and with a thoracoplasty, in addition to a pneumonectomy. As could be expected, nearly all patients of the asthmatic group are localized in the left part of the diagram.

CONCLUSIONS AND DISCUSSION

By surveying the literature on pulmonary function in patients who underwent a pneumonectomy, it is evident that the character of the data published

makes a comparison difficult for one or more of the following reasons: the time interval between pneumonectomy and follow-up is short, the data and conclusions concern small groups of patients, the data are incomplete or are only given as relative figures, the methods used differ in some extent, and the data concern nonhomogeneous groups as to the underlying diseases.

From the data reported in this paper the following conclusions can be drawn:

(a) As a result of the elimination of the vital capacity of the diseased lung by pneumonectomy, the VC_{fu} is significantly smaller than the VC_{bp} (Fig. 1). As for the whole material, no difference could be found between the VC of the contralateral lung before the operation and the VC at the follow-up

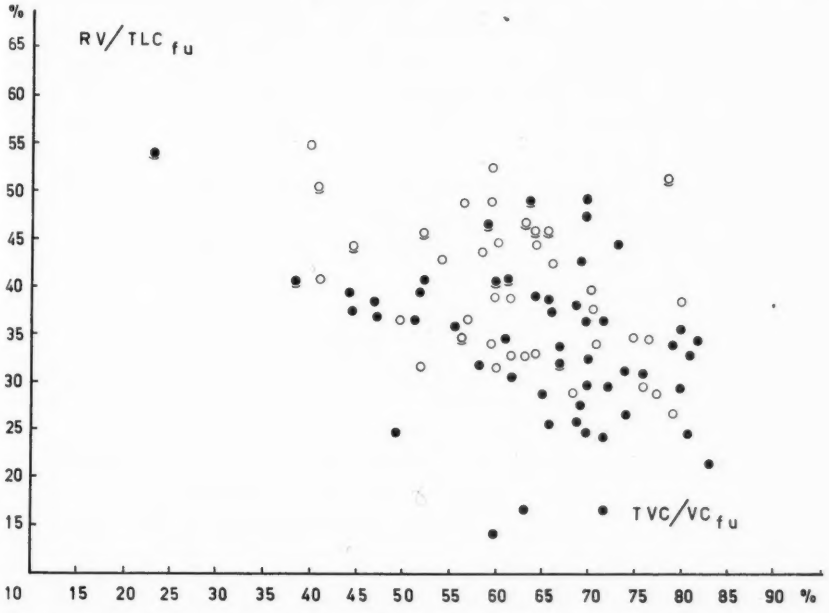

Fig. 8.—Relation between the RV/TLC ratio and the TVC/VC ratio at the follow-up examination.

study (Fig. 2). If, however, the material is divided into patients without (Group I) and with a thoracoplasty (Group II), a significant difference in this respect could be demonstrated between the two groups (Table IV). In Group I, the VC of the remaining lung was slightly increased in the period bp/fu; in Group II, on the contrary, the VC of the contralateral lung was decreased in that period. This decrease proved to be due mainly to the data of the patients who underwent their thoracoplasty after the pneumonectomy. Therefore, it seemed likely that thoracoplasties have an unfavorable influence on the vital capacity of the remaining lung. From the data of Table V, it is evident that the condition of the contralateral lung before the pneumonectomy and the

adaptability of this lung after the operation also accounts for a difference between the VC_{br} and the VC_{fu}. An increase of the VC of the remaining lung after the operation was found only when the participation of the resected lung in the total pulmonary function was relatively great (27 to 45 per cent). If, on the contrary, the function of the diseased lung was small (15 to 26 per cent), the remaining lung was apparently already partly adapted to the pathologic intrathoracic conditions before the operation and no change of the VC of that lung occurred. In the group wherein the participation of the resected lung in the VC ranged from 0 to 14 per cent, the difference between the expected loss and the actual loss was partly due to the influence of the thoracoplasty.

(b) The absolute values of the residual volume before the pneumonectomy and at the discharge from the hospital are about the same (Fig. 6). From this and from the fact that the RV of the diseased lung is eliminated by pneumonectomy it is evident that the RV of the remaining lung has increased after the operation; this holds true for both groups. The extent of the increase of the RV of the remaining lung could not be measured, because the unilateral determination of the RV was not yet at our disposal at that time. In the period between the discharge and the follow-up studies, an increase in the RV of the remaining lung was found, both in patients with and without a thoracoplasty (Fig. 6). So, in our material, an increase of the RV could not be prevented by a thoracoplasty.

(c) The TLC of the remaining lung in the postoperative period is gradually increased and this increase is for the greater part due to the change of the residual volume (Fig. 6). If the increase of the TLC is a measure of the distention of the remaining lung tissue, the distention was in most of the cases not serious and was not prevented by thoracoplasty.

(d) Many investigators have shown that, after pneumonectomy, the RV/TLC ratio, compared with the normal calculated ratio, was increased.[2, 4, 5, 6, 8, 13] Hirdes and Bosch,[9] on the other hand, found that in patients who underwent a pneumonectomy without a thoracoplasty, a decrease of the RV/TLC ratio occurred. From their data, it can be calculated that the absolute value of the RV did not change but that the decrease of the RV/TLC ratio was only a relative one due to the increase of the VC. In our material, the increase of the RV/TLC ratio was due mainly to a decrease of the VC as a consequence of the resection and later on preponderantly to an increase of the RV of the remaining lung. This holds true whether thoracoplasty was performed or not. In our material, the change of the RV/TLC ratio cannot be explained by the aging of the patients. In Group II, the RV/TLC ratio was significantly greater than in Group I, both before and after the pneumonectomy (Table VII). It could not be proved, but it seemed likely, that this difference is caused preponderantly by the presence of the thoracoplasty.

(e) Although the RV/TLC ratios of both groups differed at the time of the follow-up study, the FRC^r/TLC_{fu} ratios were almost equal (Table VI).

This was due to a difference of the ERV_{fu} of both groups. During hyperventilation, on the contrary, the FRC^h/TLC_{fu} ratio of Group II differed significantly from the FRC^h/TLC_{fu} of Group I. These findings suggest that, in rest, the efficiency of the ventilation in both groups was almost equal while, during hyperventilation, the ventilation of the patients with a thoracoplasty was less efficient.

(f) Most of the authors found the TVC/VC ratio unchanged after pneumonectomy. That means that the TVC changed in the same proportion as the VC. In our two groups, the TVC/VC ratios were identical before the operation and at the follow-up study. For judging the expiratory capacity, however, the TVC/TLC ratio proved to be a far better criterion than the TVC/VC ratio, because the changes of the RV are now taken into account. In both groups of patients, a significant difference between the TVC/TLC_{bp} and TVC/TLC_{fu} ratios was present. This difference points to the fact that a qualitative loss of the pulmonary function really occurred in both groups. Moreover, it could be proved that the expiratory capacity of patients with a thoracoplasty was significantly worse than that of the patients without a thoracoplasty.

(g) Van Veen and Hirdes[19] found a rather good negative correlation between the TVC/VC and the RV/TLC ratio in a large number of patients without asthmatic symptoms in several lung diseases. In our material, an evident negative correlation between the TVC/TLC and the RV/TLC ratio was demonstrable, even when patients with asthmatic symptoms were included.

From the data of this paper, it is concluded that thoracoplasty neither prevented distention nor increased the efficiency of ventilation or the expiratory capacity. This is in agreement with the findings of Gaensler and Strieder.[7]

Whether, at the time of the follow-up study, the remaining lung was in a stabilized condition or if, in addition to the aging process, progressive changes were taking place, could not be established. In order to gain any further insight into the fate of the remaining lung, it is of utmost importance to make repeated examinations of the pulmonary function at regular intervals.

The conclusions drawn in this paper are based on the functional data of a relative nonhomogeneous group of patients. Although the results are based on the data of a relative large number of patients, the reliability of the conclusions is only relative, because it is extremely difficult to take into account all factors influencing pulmonary function.

SUMMARY

1. The results of the pulmonary function tests in 90 patients who underwent pneumonectomy for pulmonary tuberculosis are presented. The data at the time of the follow-up study (average of 52 months after the operation) are compared with those obtained before the pneumonectomy and with the data of the pulmonary function 11 months after the operation. The patients are

divided into two groups: those without a thoracoplasty (Group I) and those who underwent a thoracoplasty before, during, or after the pneumonectomy (Group II).

2. The *vital capacity* showed a significant decrease after pneumonectomy, which corresponded well with the expected loss, as could be calculated from the bronchospirometric data. The presence of a thoracoplasty, as well as the quantity of functioning lung tissue removed, influenced the VC of the remaining lung at the follow-up study.

3. The absolute value of the *residual volume* was not changed by the operation as judged by the early postoperative study. At the time of the late follow-up study, the RV had increased in both those with and without a thoracoplasty. In the period between the pneumonectomy and the discharge from the hospital, the increase of the RV/TLC ratio was due mainly to a decrease of the VC, whereas the RV was unchanged. In the period after the discharge, the further rise of the RV/TLC ratio occurred because of an increase of the RV in the face of a nearly stable VC. The enlargement of the RV/TLC ratio is not explainable by age changes alone.

4. The *functional residual capacity* at rest showed no significant difference in the two groups. This was not true for their subdivisions—RV and ERV. During maximal hyperventilation (MBC), the FRC showed a significant increase in Group II, while in Group I a rise of the expiratory level could hardly be demonstrated.

5. The *timed vital capacity* in relation to the VC (TVC/VC ratio) was unaffected by pneumonectomy. The TVC/TLC ratio proved to be a better criterion for judging the changes of the expiratory capacity than the TVC/VC ratio. The pneumonectomy caused a significant change in the TVC/TLC ratio in both groups of patients.

6. A clear negative correlation between the RV and the TVC could be demonstrated both in patients with and without a thoracoplasty.

7. The presence of a thoracoplasty in patients who underwent a pneumonectomy proved to have no preventive influence on the distention of the remaining lung tissue and affected the efficiency of the pulmonary function in an unfavorable way.

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THE PATTERNS AND DYNAMICS OF TRACHEOBRONCHIAL DEFORMITY IN PULMONARY TUBERCULOSIS

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MANY conditions of disease in which the process of healing results in deformity of structure with great impairment of function are well recognized. Such instances as the stenosed mitral valve after acute rheumatism, the obstructed duodenum after peptic ulcer, the grotesque appearance after facial burns, and the old rigid thick-walled tuberculous cavity resisting closure, come to mind. In these, what might be called "errors of healing," the process of scar replacement and the ever-present tendency of scar tissue to contract, are the governing factors. It is as if the healing process overshoots the mark, and instead of restoring the status quo ante, actually brings about new and important disability.

These errors of healing are well exemplified in disease of the hollow organs and passages of the body. In the case of the tracheobronchial passages, healing errors are extremely frequent, and result not only from intrinsic disease of the air passages themselves, but also from disease of the pulmonary parenchyma to which they provide access. They are particularly prevalent in pulmonary tuberculosis, a disease which, by reason of its long duration and great destructive power, causes the most serious deformities of the bronchopulmonary system.

It should be emphasized that much of this tracheobronchial deformation cannot be detected in the routine x-ray studies of the chest, and can only be exposed by bronchography. It is only in the past few years that bronchography has become freely used in pulmonary tuberculosis, because of real or imagined dangers due to the effects of the heavy iodized oils, and their undesirably prolonged staining of the chest roentgenogram. The advent of the water-soluble, rapid-clearing bronchographic media, such as dionosil, has made bronchography in pulmonary tuberculosis practical and safe. Indeed, it might be said that bronchography now should be just as important a part of case evaluation before surgery in pulmonary tuberculosis as it has so long been in bronchiectasis. With the modern precise resection techniques, a careful mapping of the bronchpulmonary system is essential so that diseased segments can be removed and healthy segments preserved.

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In recent years, almost every case of pulmonary tuberculosis coming for surgical conference from the Royal Edward Laurentian Hospital, Ste. Agathe, and the Grace Dart Hospital, Montreal, has been subjected to bronchography. Some four hundred of these bronchograms have been reviewed, and this essay is an attempt to classify, correlate and, to some extent, explain the patterns of deformity found.

It should be pointed out, to begin with, that secondary bronchiectasis, although frequent, is not a constant sequel to pulmonary tuberculosis, even extensive pulmonary tuberculosis. Of the cases studied, bronchiectasis was found in approximately 50 per cent. With regard to extent of disease, 55 per cent of the whole group were in the moderately-advanced, and 35 per cent in the far-advanced group.

In general, deformities of the tracheobronchial passages may be classified as (1) enlargement or dilatation of the lumen (bronchiectasis), (2) reduction or obliteration of the lumen (stricture), (3) abnormal curvature, and (4) abnormal angulation.

TABLE I. FOUR-HUNDRED BRONCHOGRAMS

Duration of Pulmonary Tuberculosis		
1. Longer than 2 years	196	49%
2. Shorter than 2 years	204	51%
Total	400	100%
Extent of Pulmonary Tuberculosis		
1. Minimal disease	39	9.75%
2. Moderately-advanced disease	220	55.00%
3. Far-advanced disease	141	35.25%
Total	400	100.00%

TABLE II. RIGHT LUNG DISEASE

1. Bronchograms of the right lung	Normal	65	21.81%
	Abnormal	233	79.19%
		298	100.00%
2. Bronchiectasis and shrinkage of RUL		147	49.33% of total or 63.09% of abnormal
3. Bronchiectasis without shrinkage of RUL		4	1.71% of abnormal
4. Shrinkage without bronchiectasis of RUL		22	9.44% of abnormal
5. Upward deviation of RUL bronchus		27	11.59% of abnormal
6. Abnormal angulation of right main-stem bronchus		7	3.00% of abnormal
7. Cavity present at the time of bronchogram		14	6.00% of abnormal

Tracheobronchial alterations may occur in different combinations and degrees, varying from mild, limited, or localized types, to extensive, generalized deformity. For example, abnormal curvature of the trachea, convex to the side of the lesion, with secondary bronchiectasis of a diseased upper lobe is a very frequent combination (Fig. 1). Less frequently, stricture or obliteration of a lobar bronchus with atelectasis and/or bronchiectasis of the lobe is seen.

The most interesting and important alterations in pulmonary tuberculosis, however, are found as part of a general deformation of the tracheobronchial skeleton, due to marked upward traction displacement of the hilar structures (Fig. 2).

It is well known that shrinkage of the diseased upper lung with elevation of the hilus is a common finding in pulmonary tuberculosis. Indeed, it has been generally regarded as a somewhat desirable thing, in that it implies or

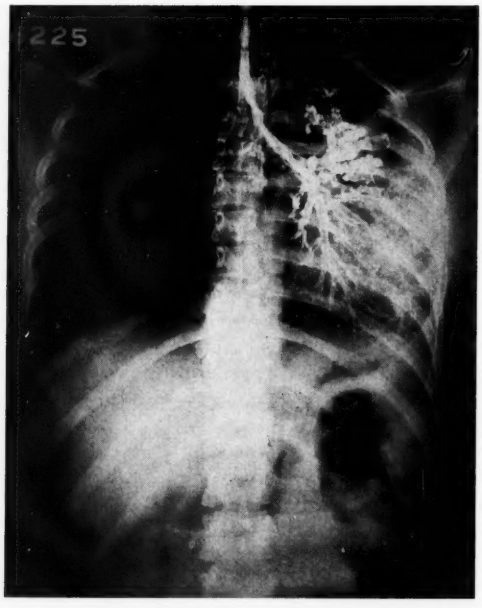

Fig. 1.

Fig. 2.

TABLE III. LEFT LUNG DISEASE

1. Bronchograms of the left lung	Normal	61	25.20%
	Abnormal	181	74.80%
	Total	242	100.00%
2. Bronchiectasis and shrinkage of LUL		131	54.13% of total or
3. Bronchietasis without shrinkage		1	72.38% of abnormal
4. Shrinkage without bronchiectasis		0	
5. Abnormal curvature of the left main-stem bronchus and abnormal angulation, junction left main-stem bronchus and left lower lobe bronchus		81	61.83% of diseased LUL; 44.75% of abnormal
6. Cavity present at time of bronchogram		19	10.05% of abnormal

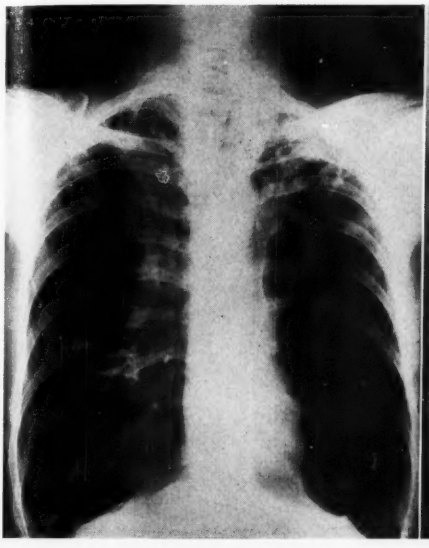

Fig. 3.

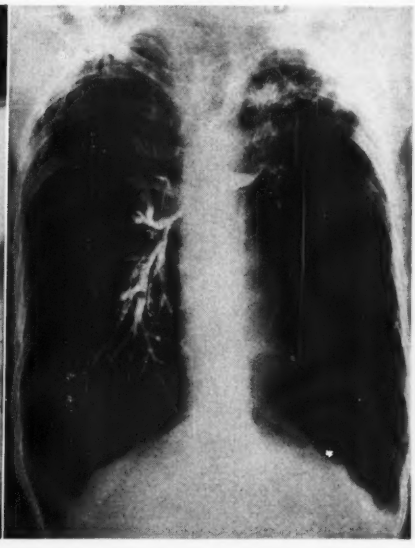

Fig. 4.

is thought to imply sound fibrous healing of the lesion. Not enough attention has been paid, however, to the deleterious effect on function of this upward pull on the hilus. This is no mere local shrinkage of organs, but a serious structural change of the bronchopulmonary alignment on the side in question or, in the case of bilateral disease, of the whole bronchopulmonary framework. So serious can this be that the ultimate result of an original lesion of the upper one third of the left lung, for example, may be virtually complete functional loss of the entire left lung, the upper lung being destroyed by tuberculosis and bronchiectasis, and the lower lung by emphysema (Figs. 3 and 4).

Such a condition is exemplified in Fig. 3. The bronchogram shown in Fig. 4, of the same case, illustrates the typical deformity resulting from upward traction displacement of the left hilus. The abnormal curvature of the

left main-stem bronchus with pooling of the radiopaque liquid, and the abnormal angulation at the junction of the left main-stem and left lower lobe bronchus are the important features.

Due to anatomic differences as between the right and left sides, the effect of upward traction displacement is not as serious on the right as on the left, because the lower lung on the right is not so vulnerable.

This is explained by reference to certain differences in the anatomy of the bronchopulmonary system as between the right and left sides (Figs. 5 and 6). The left main-stem bronchus is much longer and it lies much more horizontally than the right main stem. Furthermore, it passes immediately beneath the arch of the aorta. If one regards both main stems as the arms of a lever (Fig. 5) with the fulcrum at the carina, it is clear that a relatively slight upward pull on the long left arm will have an equal effect to a much stronger pull on the short right arm.

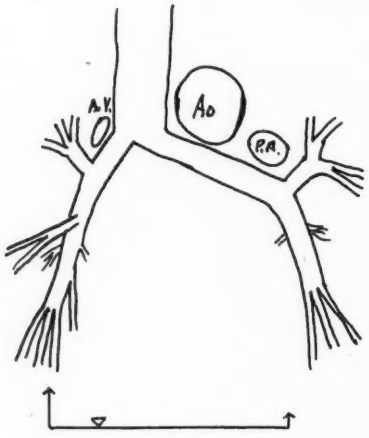

Fig. 5.

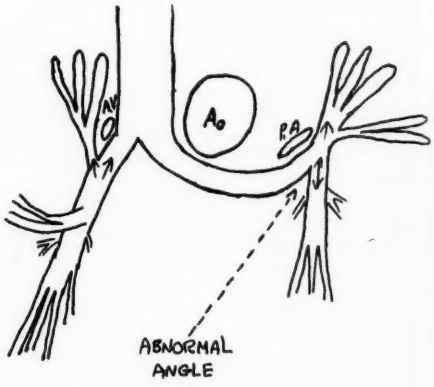

Fig. 6.

Hence, to begin with, upward traction displacement of the hilus is always more pronounced, and thus more serious on the left side. Normally, there is a gentle curving of the medial wall of the left main stem as it runs into the left lower lobe bronchus. When the left hilus is pulled up, the long left main stem winds itself around the arch of the aorta, into an abnormal curve, concave upward, and what was originally a gentle curve at the junction of the left main-stem and the left lower lobe bronchus now becomes an abnormal angle, which in long-standing cases may be as acute as 50 degrees. In the group under review, this angle varied between 50 and 130 degrees. This abnormal angulation obviously changes greatly the luminal axis of the left bronchial tree, and has a pronounced disturbing effect on its function.

In addition to the bronchus, all the structures of the hilus are involved in this change. The left pulmonary artery normally lies at first in front of the left main-stem bronchus, then winds up over the main stem before this branches into the upper and lower lobe bronchi. The artery then hooks

around, as it were, behind the upper lobe bronchial arborization to emerge underneath it, and finally comes to lie in front of the lower lobe bronchus. When the left upper lobe arborization is pulled up, the artery is sharply kinked at the point where it crosses above the main stem and reduction in lumen with reduced blood flow to the left lower lobe occurs. Fig. 7 is a demonstration at autopsy of such an occurrence. Note the marked reduction in caliber of the pulmonary artery to the left lower lobe.

On the right side, a different picture presents itself. Here, because of the short, relatively vertical main stem, the upward displacement is practically confined to the more horizontal right upper lobe bronchus, which becomes more or less sharply angulated upward to lie more in the axis of the trachea. This is usually accompanied by bronchiectasis of the right upper lobe. A further stage of the deformity, and a typical occurrence on the right side, is obliteration of the lumen of the right upper lobe bronchus, the right

Fig. 7.

upper lobe itself coming to lie, a shrunken atelectatic remnant, against the mediastinum (Fig. 8). This is reminiscent of the well-known middle lobe syndrome.

In a few instances of long-standing disease with great shrinkage of the right upper lobe, we have found a slight degree of upward displacement of the right main stem, but this is unusual and never to the same extent as on the left side. Furthermore, there is no tendency toward curvature on the right because the small compressible azygos vein does not offer the same resistance as the large thick-walled aorta on the left.

It should be noted here, therefore, that the ultimate effect of upward traction displacement on the right side is exerted on the right upper lobar

hilus, while on the left side it is exerted on the entire pulmonary hilus. This may explain the greater vulnerability of the left lung and the fact that the right lower and middle lobes are not so liable to become emphysematous.

The effects of these changes in lumen and alignment are, we believe, responsible for profound disturbances of function. The first and perhaps the most obvious effect is fixation or relative fixation of the hilar area by this strong upward pull, acting as a taut guy rope on the hilar bronchial branching. Thus, the normal up and down movement of the hilus on expiration and inspiration, respectively, is greatly hindered or prevented. This is apt to restrict ventilation to a marked degree, since it acts in direct opposition to the normal downward pull of the diaphragm. In this way, it hinders the easy downward expansion of the parenchyma, as the bronchial tree moves downward and to some extent inward on inspiration.

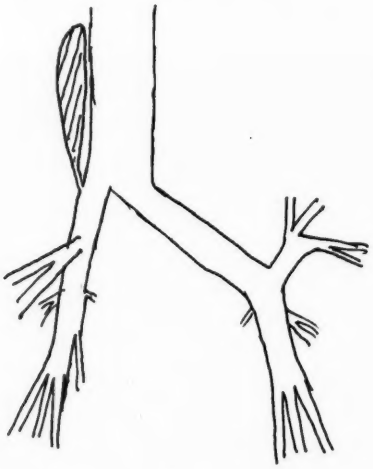

Fig. 8.

Another obvious effect is impaired gas exchange, due to reduction in lumen of the left pulmonary artery, and hence lessened blood supply, particularly to the left lower lobe. Incidentally, some observers believe that emphysema can develop as a direct result of reduced pulmonary artery blood flow. The effects of stimulation due to traction on the elements of the hilar nerve plexus could be significant, but the importance of this can only be guessed at.

Probably the most far-reaching effects, however, are due to the altered caliber and alignment of the air passages with resulting changes in the dynamics of bronchopulmonary air flow. Currents of gas like rays of light tend to travel in straight lines. When a current of air comes up against an obstruction or partial obstruction in a bronchial passage, such as a stricture or a sharp angulation, or a pronounced curve, there is hindrance to air flow. The result of this is deflection of the stream, secondary currents and eddies,

and perhaps most important, reverse currents of flow. In quiet respiration this may not interfere too much with function. In deep forcible breathing, however, there may be considerable disturbance and this is first and chiefly felt in interference with air exit. In a markedly impaired lumen, there is also interference with air entry, but the first disturbance is difficulty in emptying rather than filling the lungs. The normal dilatation of the bronchial passages on inspiration, and their corresponding contraction on expiration accentuates the obstructive effect of abnormal angulation or stricture, particularly with respect to air exit. The ultimate effect is the phenomenon known as "air trapping."

The most extreme conditions of air flow occur in cough, and particularly in forcible, violent, prolonged and repeated cough, and under these conditions the most intense disturbance of bronchopulmonary dynamics is apt to occur. Reverse currents of air flow, now become reverse shock waves, and instead of simple air trapping, there occurs a powerful abnormal backward thrust on the walls of the distal bronchi and alveoli.

Rahn has estimated that in a particular phase of cough the air is traveling at speeds in the realm of 200 to 300 miles per hour. Beck has reported air flow rates of 12 L. per second at the mouth, at the moment of glottic opening in cough, and says that the air speeds reached here are sometimes higher than the speed of sound, namely, 732 miles per hour. Beck also reports that during the glottic closure phase of cough, the intrathoracic pressure in normal subjects ranges between 80 and 110 mm. Hg, while in patients with bronchopulmonary disease, the pressure may be increased over 120 mm. Hg. We have measured the intrabronchial pressures during cough, and in forcible cough have registered pressures as high as 180 mm. Hg in the lower lobe bronchi. These figures give an idea of the magnitude of the forces that are operative in the bronchopulmonary system during cough. It is understandable, therefore, that a possible result of the long-continued action of these forces may be collapse or blow out of the walls of bronchi or alveoli, encouraging or causing, in the one case, bronchiectasis and, in the other, emphysema. The association of emphysema with the cough-producing propensities of chronic bronchitis, and of bronchiectasis with a history of whooping cough in childhood is, perhaps, significant.

Not enough emphasis, we believe, has been placed on violent uncontrolled cough as an injurious factor leading to extensive lung destruction. In patients with lung disease, cough is apt to be more prevalent and forcible in order to get rid of abnormal secretions or because of mucosal irritation. Again, if the bronchial passage is deformed, it becomes more difficult to move secretions through it. Moreover, people differ widely with respect to the control which they exercise over the function of cough. Coughing, like scratching, is a physiologic mechanism designed to remove an irritant to a body surface. It is always available, but is intended to be used only when needed, and then nicely graded to the extent of the need. One cough, like one scratch, borrows another, and uncontrolled coughing or scratching can result in serious damage to the structures acted upon.

Let us again consider (see Figs. 5 and 6) the altered structure of the bronchopulmonary skeleton in upward traction displacement of the hilus, and the effect of these alterations on function. Normally the expired air coming up from the lower lobe flows easily into the left main-stem bronchus and thence into the trachea. When the left hilus is pulled up, however, the air current from the left lower lobe will tend to flow into the dead end of the diseased upper lobe because the left upper lobe bronchus is now in more or less direct line with the left lower lobe bronchus, whereas the left main stem has actually taken a turn in the opposite direction. This obviously impedes the exit of air from the left lower lobe. In cough, there is added a powerful abnormal backward thrust on the alveolar walls of the left lower lobe, and a forward thrust on the already diseased and weakened bronchi of the left upper lobe.

In upward traction displacement of the right hilus, there is a similar though less marked abnormal forward thrust on the diseased bronchi of the right upper lobe, but there is obviously never the same degree of obstruction to air exit and hence not the same degree of abnormal backward thrust on the alveolar walls of the right lower lobe.

It is difficult, except in advanced cases, to detect the presence of emphysema on the chest roentgenogram, but we have had occasion to remark on the relative frequency of emphysema of the left lower lobe in pulmonary tuberculosis.

The finding in chronic phthisis of upper lobe shrinkage with lower lobe emphysema is common. The emphysema is usually designated as compensatory. It is assumed that the uninvolved lower lung dilates to fill the space vacated by the shrunken diseased upper lung. If it were merely a need to fill space, however, one would expect the diaphragm to move upward in order to reduce the need, as it usually does after lobectomy on the side in question. Furthermore, one would expect not the lower lung but the mid-lung to dilate since this is the part contiguous to the diseased portion and hence should be the part first and chiefly subjected to this presumed upward call for space-filling tissue. Neither of these things happen, however. In fact, the diaphragm moves downward instead of upward, and the part of the lung that becomes emphysematous is the lower and not the mid-zone. This suggests that the so-called compensatory emphysema is the result of a different and possibly much more complex mechanism than the one usually supposed. It is highly possible that the emphysema is obstructive rather than compensatory, and it could develop as the result of upward traction displacement of the hilar structures and poorly controlled cough.

In the course of this study we found it convenient to recognize a left lung syndrome, and a right upper lobe syndrome (Tables IV and V).

TABLE IV. LEFT LUNG SYNDROME

1. Upward traction displacement of left hilus
2. Abnormal curvature left main-stem bronchus
3. Abnormal angulation at junction of left main-stem bronchus and lower left lobe bronchus
4. Bronchiectasis, left upper lobe
5. Emphysema, left lower lobe (final stage)

TABLE V. RIGHT UPPER LOBE SYNDROME

Stage 1
1. Abnormal angulation or partial occlusion right upper lobe bronchus
2. Bronchiectasis, right upper lobe.

Stage 2
1. Marked abnormal angulation and complete or almost complete occlusion right upper lobe bronchus
2. Atelectasis, right upper lobe

SUMMARY

We believe the most significant finding of this study is the evidence pointing to the undesirable effects of upward displacement of the hilus, particularly on the left side, in chronic pulmonary tuberculosis. This points up the need for vigilance, especially in tuberculosis of the left upper lobe, to anticipate and if possible prevent the destructive effects of this displacement.

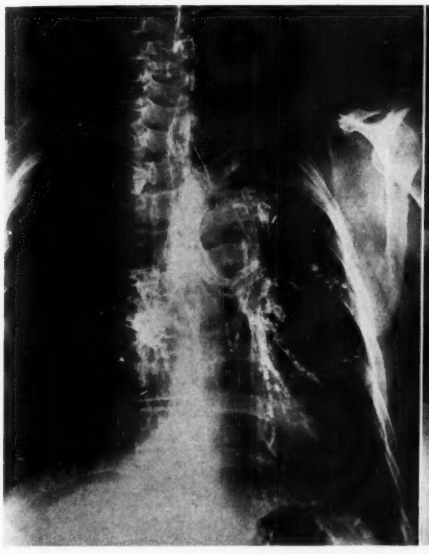

Fig. 9.

Fig. 10.

What are the chances for preventing or interrupting such a development?

Prevention is obviously best served by prompt treatment of the tuberculosis in order to diminish the amount of contracting scar tissue. We suspect also that the reduction or elimination of useless forcible cough is important.

In order to interrupt the deforming process, it would seem as if the only logical way of proceeding would be to remove the contracting mass of fibrous tissue in the left upper lung as soon as it is observed to be causing abnormal curvature of the left main-stem bronchus, and abnormal angulation at the junction of the left main stem and left lower lobe bronchi. This would be a

new indication for removing an area of disease, even apparently healed disease in the left upper lung. In cases in which, for some reason, resection of the left upper lung is not practical, an alternative might be found in upper thoracoplasty with apicolysis, pushing the contracting mass down to the level of the hilus where its upward pull is neutralized.

When deformity is already far advanced and the left lung syndrome well established, there is less hope for useful remedial measures. However (Figs. 9 and 10) in some cases of this kind, resection of the left upper lobe with upper thoracoplasty does seem to correct, at least in part, the abnormal curvatures and angulation, and in this way tend to interrupt the harmful series of events leading to loss of function of the left lower lobe.

The authors wish to acknowledge the kind cooperation of the Medical Director and staffs of the Royal Edward Laurentian Hospital and the Grace Dart Hospital.

THE SURGICAL TREATMENT OF AORTIC INSUFFICIENCY BY CONVERSION OF THE TRICUSPID AORTIC VALVE TO A BICUSPID VALVE

Chronic Experimental Studies

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IN PREVIOUS communications,[1, 2] a method of aortic valvuloplasty was described for the surgical treatment of aortic insufficiency. The method consists of total excision of the noncoronary aortic cusp with a corresponding triangular segment of the base of the aorta (Fig. 1). By suture repair, the normal

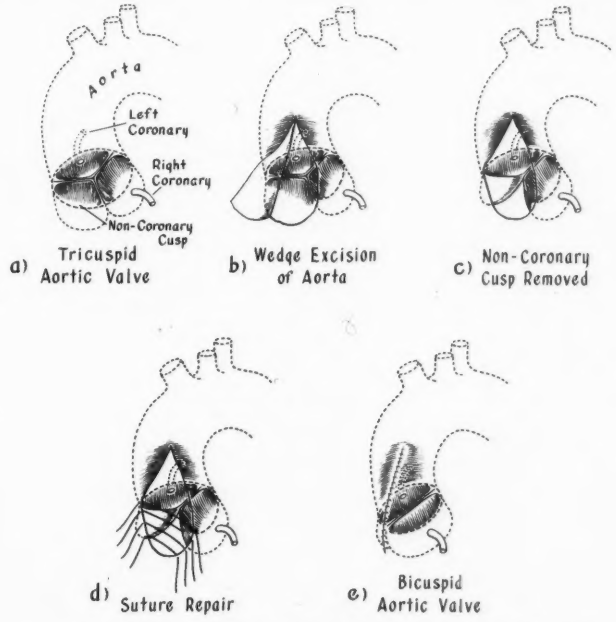

Fig. 1.—Conversion of tricuspid aortic valve to bicuspid valve.

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Fig. 2.

Fig. 3.

Fig. 4.

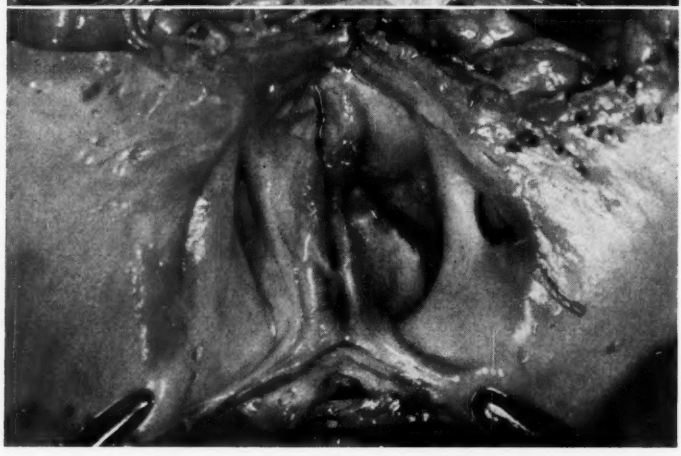


Fig. 2.—An autopsy specimen from a patient dying of aortic regurgitation shows the insufficient aortic valve viewed from above and posteriorly. Note the distortion, thickening, and shortening of the aortic cusps. The central defect causing aortic regurgitation is evident. The loss of cusp substance precludes complete coaptation. Dilatation of the aortic ring is not a conspicuous feature of this specimen.

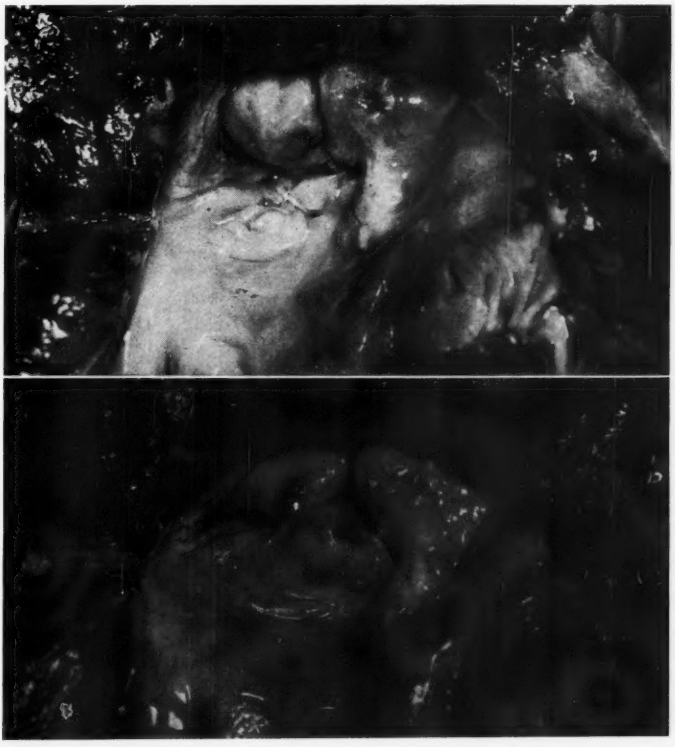
Fig. 3.—Insufficient aortic valve viewed from below, the ventricular side, showing the central defect causing regurgitation. A partial rim of calcification is noted.

Fig. 4.—The incompetent aortic valve seen in Figs. 4 and 5 is viewed from above following plastic revision with total excision of the noncoronary aortic cusp. The valve is now bicuspid. The circumference has been reduced by one third. The two remaining coronary cusps appear to coapt well. The left coronary ostium is visualized to the left.

tricuspid aortic valve is converted to a bicuspid valve and the aortic circumference is reduced by one third. In effect, a severe degree of aortic insufficiency is produced and then surgically corrected by the valvuloplasty. Operative procedures were performed under direct vision with cardiac bypass employing an extracorporeal pump-oxygenator and elective cardiac arrest.

Short-term animal survival studies were described as well as functional evaluation in terms of pre- and postoperative arterial pulse patterns, transventricular and systemic pressure determinations, and retrograde aortography.

A.



B.

Fig. 5.—*A*, The incompetent aortic valve seen in Figs. 4 and 5 is viewed from below, ventricular side, following plastic revision. The noncoronary aortic cusp has been excised. The newly formed bicuspid valve appears to coapt well. Sutures in the line of repair are evident.

B, The aortic valve again viewed from the ventricular side. The noncoronary aortic cusp remains in situ while the aortic wall has been reduced by one third. The resultant coaptation appears less effective than in *A*, the noncoronary cusp seemingly herniating below the coronary cusps.

Phases of systole were measured. No significant degrees of aortic stenosis nor aortic insufficiency were found in surviving animals. Pathologic studies as evidenced in Figs. 2, 3, 4, 5*A*, 6 support the clinical application of the principle.

Accordingly, it was felt that functional studies in chronic survival animals would be of interest and valuable and these form the basis of this report.

MATERIALS AND METHODS

Eleven chronic survivors with surgically produced bicuspid aortic valves were studied 26 to 405 days postoperative. The animals were anesthetized with intravenous Surital (thiamylal sodium), 75-100 mg. A No. 18 gauge needle was inserted percutaneously into the femoral artery. A No. 13 to 15 gauge

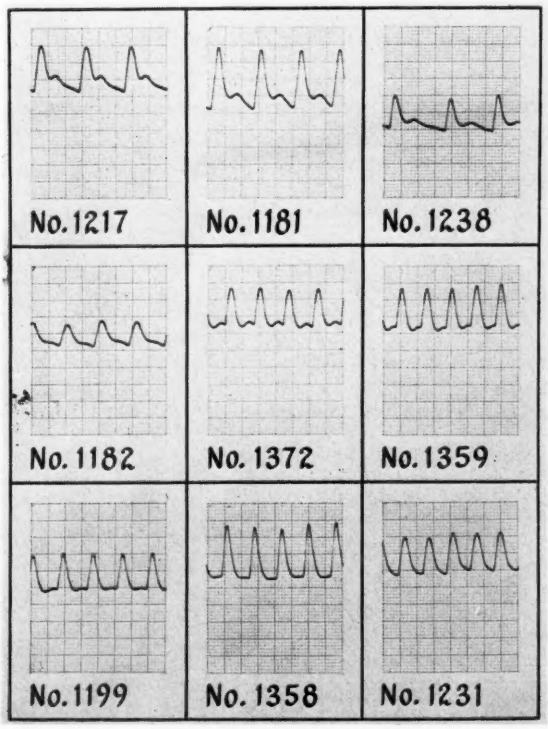


Fig. 6.—Conversion of tricuspid aortic valve to bicuspid valve. Analysis of femoral pulse contours. Chronic studies 26-317 days postoperative.

TABLE I. CONVERSION OF TRICUSPID AORTIC VALVE TO BICUSPID VALVE CHRONIC STUDIES 26-317 DAYS POSTOPERATIVE

ANIMAL NO.	DAYS POSTOP.	DICROTIC FEMORAL CURVE	B.P. (FEMORAL)	PULSE PR. (FEMORAL)	MAXIMAL EJECTION PERIOD	REDUCED EJECTION PERIOD	TOTAL SYSTOLE
1182	317	+	155/85	70	0.08	0.16	0.24
1181	309	+	165/80	85	0.08	0.14	0.22
1199	305	+	200/110	90	0.08	0.14	0.22
1217	289	+	175/105	70	0.08	0.12	0.20
1193	282	+	150/105	45	0.06	0.10	0.16
1231	272	–	150/95	55	0.06	Absent Dicrotic Notch	
1238	259	+	200/100	100	0.06	0.12	0.18
1330	173	+	135/100	35	0.06	0.12	0.18
1358	167	+	165/90	75	0.06	0.16	0.22
1359	26	+	85/55	30	0.06	0.12	0.18
1372	60	+	165/110	55	0.06	0.12	0.18

needle was passed percutaneously into the left ventricle at the point of maximal impulse, usually the fifth intercostal interspace. A polyethylene catheter of appropriate size was threaded through this needle into the ventricle and then through the bicuspid aortic valve into the central aorta. Proper connections were made to Statham strain gauges P23A and a Sanborn Poly-Viso recorder with recording speed of 25 mm. per second.

RESULTS

Analyses of femoral artery pulse curves (Table I and Fig. 6) show the presence of dicrotic notches in ten of eleven studies. Several animals showed broadened pulse pressures. Significant alterations in the duration of systolic ejection phases were not found.

Continuous pressure recordings from the central aorta to the left ventricle (Table II and Fig. 7) show a minor systolic gradient in 4 animals. End diastolic pressures were slightly elevated in 2 animals.

TABLE II. CONVERSION OF TRICUSPID AORTIC VALVE TO BICUSPID VALVE
CENTRAL AORTIC AND LEFT VENTRICULAR PRESSURES
CHRONIC STUDIES 80-405 DAYS POSTOPERATIVE

ANIMAL NO.	DAYS POSTOP.	PRESSURE MM. Hg						
		CENTRAL AORTA	LEFT VENTRICLE	SYSTOLIC AORTIC VALVE GRADIENT	END DIASTOLIC LEFT VENTRICLE	CENTRAL AORTIC PULSE	MEAN	
							CENTRAL AORTA	LEFT VENTRICLE
1182	405	125/100	125/0-13	0	0-13	25	115	55
1181	344	135/100	135/0	0	0-5	35	100	83
1199	344	100/75	140/0	40	0	25	90	55
1217	374	125/95	125/5	0	5	30	100	45
1193	321	110/75	120/–5	10	–5	35	100	65
1231	317	100/80	135/0-15	35	0-15	20	90	50
1238	317	110/85	125/0	15	0	25	85	50
1330	211	80/45	80/0-5	0	0-5	40	55	33
1358	221	160/130	165/0	5	0	30	135	100
1359	80	100/80	95/0-5	–5	0-5	20	90	50
1372	114	115/90	115/0	0	0	25	105	50

Since the completion of these studies (90+ days) the animals continue to be well, displaying normal activities for caged subjects.

DISCUSSION

Acute and chronic studies demonstrate that dogs tolerate plastic revision of the aortic tricuspid valve to a bicuspid valve. Pathologic studies as shown in the manuscript support the feasibility of this concept and method for clinical application in the surgical treatment of aortic insufficiency. More recent studies by us, employing hydraulic and cinephotographic studies as described by McMillan,[3] show that (1), at least in rheumatic aortic valvular insufficiency, the noncoronary aortic cusp appears to interfere with the coaptation of all three cusps and accordingly contributes to the pathogenesis of the regurgitation, and (2) the reconstructed bicuspid aortic valve in the same diseased specimens coapts remarkably well and minimizes regurgitation.

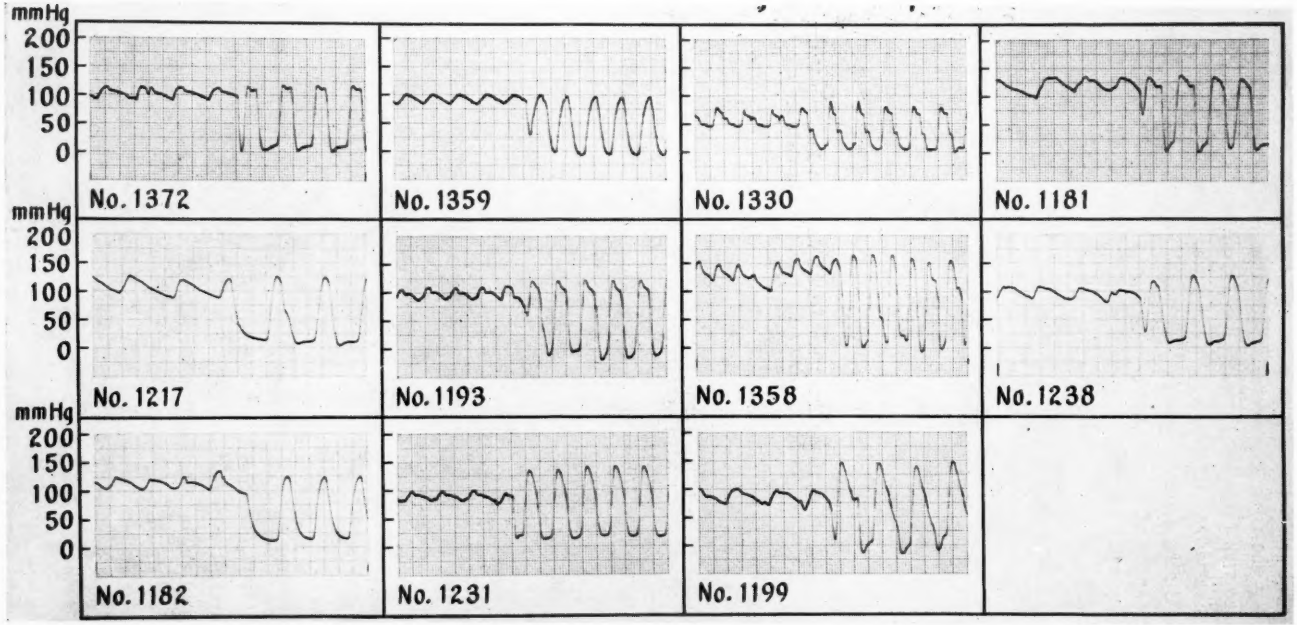


Fig. 7.—Conversion of tricuspid aortic valve to bicuspid valve. Continuous pressure curves from central aorta and left ventricle. Chronic studies 80-405 days postoperative.

SUMMARY

1. A method of aortic valvuloplasty, conversion of the tricuspid aortic valve to a bicuspid valve, is described for the surgical treatment of aortic insufficiency.

2. Chronic experimental studies demonstrate the canine tolerance for the procedure.

3. Pathologic and cinephotographic studies support the clinical application of the concept and method.

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HYPOTHERMIC ANALGESIA FOR OPEN HEART SURGERY WITH THE HEART-LUNG MACHINE

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EXTRACORPOREAL circulation is an accepted technique used to facilitate and make possible the surgical correction of formerly incurable congenital and acquired cardiovascular lesions.[1] This surgical advent has brought about certain exigencies in the field of anesthesia about which little has been reported. In the studies that have been made and reported of anesthesia during extracorporeal circulation,[2] the use of depressant anesthetics and hypnotics is advocated before and during the period of cardiopulmonary bypass.[2] Patients requiring extracorporeal circulation for correction of disease must be classed as "poor risk" and therefore do not tolerate depressant drugs or hypoxia very well. Light planes of anesthesia frequently produce arrhythmias and persistence to deeper planes many end disastrously. To facilitate surgical intervention in such patients, the use of anesthetic adjuncts, such as muscle relaxants,[3] hypothermia,[4] analgesic planes of potent anesthetics,[5] and hypnosis[6] has come into vogue. All of these have finite salutary attributes and these effects have been incorporated into one technique and utilized during the various stages of any one operation. Such a combined technique most closely approximates the "ideal" of the perfect anesthetic for these "poor risk" patients. It must be remembered, however, that one of the most critical periods of open heart surgery is that of cardiopulmonary bypass. It is during this period that paucity of drug effect is most desirable. With that end in view, an analgesic level of hypothermia was postulated. It was felt that such a state would obviate the use of depressant drugs in order to maintain satisfactory operating conditions during bypass.

In 1950, Bigelow and associates[7] pointed out that animals cooled below 28° C. entered a state of "cold narcosis" during which an anesthetic agent was no longer necessary for maintaining unconsciousness and relaxation. In 1950, McQuiston[8] showed that surgical correction of congenital heart conditions is more safely performed with the patient's temperature reduced.

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Under hypothermia, adrenal stress reactions are both reduced and delayed, and patients who are operated on under those conditions do not demonstrate the usual retention of water and salt during the early postoperative period.[9] Moderate hypothermia (30°-32° C.) lowers cerebral metabolism from 75 to 40 per cent.[10] Virtue[11] has stated that with a reduced cerebral metabolic rate, a smaller amount of anesthetic is required to obtain surgical anesthesia. Ventricular fibrillation is more difficult to treat in the hypothermic state and at esophageal temperatures of 28° C. or below, ventricular fibrillation is more apt to occur. Recently, experimental evidence has been accumulating that the cholinomimetic agent, succinylcholine, not only has fine muscle relaxing qualities but also blocks the cardiac effects of sympathetic and parasympathetic stimulation by depolarization.[12]

Two methods of producing hypothermia are now being employed.[13] The most frequently used method consists of surface cooling. The patient is first anesthetized and then immersed in an ice water bath or covered with ice bags. The other method consists of utilizing extracorporeal circulation to produce hypothermia. A method of open heart surgery utilizing extracorporeal circulation and producing analgesic level of hypothermia has been employed and found to be most satisfactory.

METHODS

The Kay-Anderson pump-oxygenator* (Fig. 1) was used for all of these operations. The apparatus is operated by one person and is capable of oxygenating and pumping 6 L. or more of blood per minute. Metabolic acidosis does not occur during cardiopulmonary bypass. Nine units of blood are used in the apparatus during the procedure. The blood is oxygenated by blowing 10 L. of 97.5 per cent oxygen and 2.5 per cent carbon dioxide across the filming screens. During the procedure, the aortic pressure is monitored and maintained at a relatively normal mean pressure. The electrocardiogram is also monitored. A thermocouple lead is placed in the lower third of the esophagus for constant temperature readings. Blood volume studies are done before and after surgery. The blood used in the machine is approximately 5 hours old and is placed in a water bath at 38° C. 1 hour prior to filling the pump-oxygenator. However, the oxygenator, which is made of stainless steel, is not warmed prior to adding the blood or during bypass. This accounts for the sustained hypothermia until the end of cardiopulmonary bypass.

In order to avoid any possible deleterious effect on the cardiovascular system, no preoperative medication is given. Cyclopropane and/or ether is used for all inductions. The younger patients are induced with open cyclopropane and the older patients with a closed circle system. Following induction, venous cutdowns are performed. After tracheal intubation, all patients are put on a semiclosed system employing nitrous oxide and oxygen and are given a slow continuous drip of succinylcholine (Anectine). The thoracotomy incision is made by one team while a separate team isolates both femoral arteries. After the femoral vessels are isolated, this team completes the opening of the left chest,

*Constructed by Corco, Incorporated, 10418 Venice Boulevard, Los Angeles 34, Calif., for Dr. Kay and Dr. Anderson.

while the other team completes the opening of the right chest. As soon as both sides of the chest are entered, manually controlled respiration is instituted and maintained until the chest is closed again. As soon as extracorporeal circulation is started and during the intracardiac repair, the drip rate of succinylcholine (Anectine) is doubled. A mixture of 40 per cent oxygen and 60 per cent helium is used to inflate the lungs gently 5 to 10 times per minute during bypass. This is done to ensure adequate oxygenation of alveolar epithelium[14] and to prevent unresolvable atelectasis.

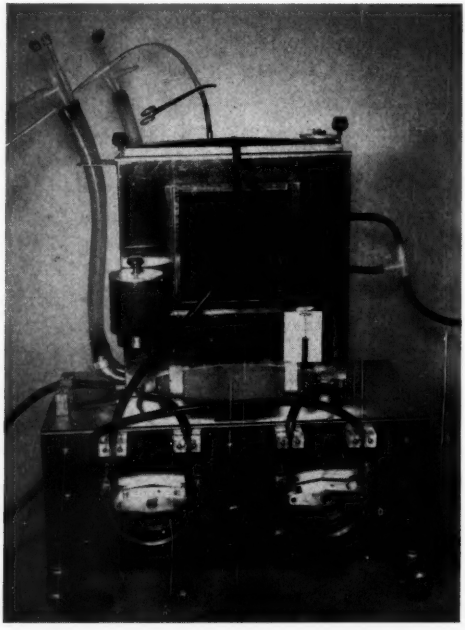

Fig. 1.—The Kay-Anderson autoclavable stainless steel heart-lung machine.

Immediately upon going on bypass, there is a sudden drop in body temperature as measured by the esophageal lead (Fig. 2). This drop continues for the next 3 minutes and levels off after 2 more minutes between 31° and 31.5° C. This temperature is sustained during the entire time that extracorporeal circulation is maintained. During this time, no attempt is made to introduce any anesthetic drug other than to keep all striated muscle paralyzed with succinylcholine. Upon completion of bypass, the succinylcholine drip is completely discontinued and 50 per cent nitrous oxide and 50 per cent oxygen is started. This mixture is maintained until completion of the surgery. Following cardiopulmonary bypass, no other drug or agent is required to keep the patient anesthetized. Also, during this period, the temperature gradually rises (Fig. 2) and at the end of surgery is only 1° to 2° C. below normal. No attempt is made to warm the patients artificially. At the completion of the operation and upon discontinuance of the nitrous oxide, all of the patients are awake.

RESULTS

Fifteen patients underwent surgical correction of their cardiac defects using this technique (Table I) and all reacted identically, with one exception. This patient had a prolonged succinylcholine effect. He was awake at the end of surgery; however, he required assisted respirations for about 30 minutes postoperatively.

In spite of the fact that preoperative drying agents were not used, respiratory secretions were not a problem. The usual cardiac slowing, seen under hypothermia, was not encountered in any of the cases.

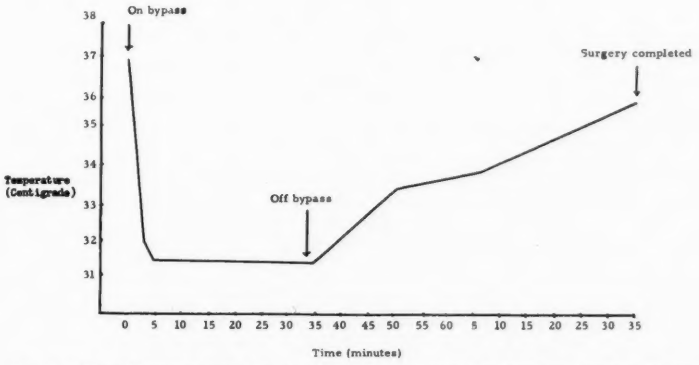

Fig. 2.—Time-Temperature Chart. Case 1—on cardiopulmonary bypass 32 minutes.

The types of surgery and the ages of the patients are shown in Table I. With the exception of Case 4, all patients did extremely well postoperatively. Case 4 died 18 hours after surgery. Although precautions were taken in an attempt to prevent cardiac tamponade, a thick clot formed around the heart and produced cardiac tamponade. The patient was alert until shortly before death. Necropsy revealed the tamponade and excellent closure of the interatrial and interventricular septal defects. Since then the pericardial sac has not been closed.

The length of time of bypass varied from 16 minutes to 42 minutes. Careful questioning of all patients revealed that no one had any recollection of any part of the surgery. All patients remembered going to sleep and the younger patients remembered only the stories they were told during the induction.

COMMENT

In planning this anesthetic technique, drugs that might have a deleterious effect on the myocardium were avoided. By warming the heparinized blood to 38° C. prior to filling the machine, and not warming the stainless steel oxygenator, an almost constant time-temperature reduction could be predicted. The temperature fall did not appear to be related to the weight of the patient or the time on bypass, after the first 5 minutes. This allowed us to arrive at levels of "Hypothermic Analgesia." At these levels (31° to 32.5° C.), no anesthetic

TABLE I. ANALYSIS OF FIRST 15 PATIENTS UNDERGOING INTRACARDIAC SURGERY UTILIZING HYPOTHERMIC ANALGESIA

PATIENT	DATE OF OPERATION	AGE (YEARS)	WEIGHT (LBS.)	DIAGNOSIS	PROCEDURE	OUTCOME
1. K. H.	12-12-57	8	70	Ventricular septal defect	Closure of defect	Lived; defect completely closed
2. J. F.	1- 9-58	8	40	Ventricular septal defects	Closure of defects	Lived; defects completely closed
3. A. V.	1-16-58	8	50	Patent ductus arteriosus and ventricular septal defects	Ligation of ductus and closure of defects	Lived; defects completely closed
4. D. L.	1-30-58	4	28	Atrial septal defect and ventricular septal defect	Closure of atrial and ventricular septal defects	Died 18 hours postop. of cardiac tamponade; both defects completely closed
5. R. C.	2- 6-58	8	70	Atrial septal defect and valvular pulmonic stenosis	Closure of atrial septal defect and pulmonary valvulotomy	Lived; defects completely treated
6. A. DeL.	2-20-58	7	50	Patent ductus arteriosus and tetralogy of Fallot	Ligation of ductus, resection of infundibular stenosis and closure of ventricular septal defect	Lived; defects cured
7. R. M.	2-27-58	2	29	Patent ductus arteriosus and ventricular septal defect	Defect closed and ductus ligated	Lived; defects completely closed
8. N. P.	3-12-58	4½	38	Ventricular septal defect	Closure of defect	Lived; defect completely closed
9. D. S.	3-20-58	4¾	39	Ventricular septal defect	Closure of defect	Lived; defect completely closed
10. S. H.	3-27-58	7	40	Patent ductus arteriosus and ventricular septal defects	Ductus ligated and defect closed	Lived; defects completely closed
11. D. S.	4- 3-58	6	45	Ventricular septal defect	Closure of defect	Lived; defect completely closed
12. R. J.	4-17-58	2¾	32	Ventricular septal defect	Closure of defect	Lived; defect completely closed
13. B. J.H.	4-28-58	8	40	Ventricular septal defect	Closure of defect	Lived; defect completely closed
14. L. A. E.	5- 7-58	3	30	Ventricular septal defect	Closure of defect	Lived; defect completely closed
15. W. T.	5-22-58	23	143	Ventricular septal defect	Closure of defect	Lived; defect completely closed

was necessary. Muscular movement of the patients was controlled and untoward cardiac reflexes did not occur. This was probably due to the succinylcholine.

SUMMARY

A new concept of hypothermic analgesia for extracorporeal open heart surgery is presented. Except for a muscle relaxant, no other anesthetic adjunct was required to maintain surgical anesthesia during bypass. Fifteen cases are reported with only one mortality and the technique is described.

ADDENDUM

The authors now use a median sternotomy instead of a bilateral anterior thoracotomy.

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THE EFFECT OF PROLONGED VENO-ARTERIAL PUMPING ON THE CIRCULATION OF THE DOG

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*With the Technical Assistance of Carl E. Hewson.***

INTRODUCTION

THE VOLUME work of the heart may be reduced by diverting part of the venous return and pumping it into the aorta. Such veno-arterial shunting may prove to be beneficial for days following myocardial infarction or pulmonary embolism. The purpose of this study is to determine the long-term effects of pumping a large proportion of the venous return into the aorta on cardiac output, regional oxygen supply, clotting mechanism, and acid-base balance.

METHODS

The technique and equipment for veno-arterial shunting are described in detail in a separate report. Blood flows by gravity through a cannula with tip placed at the junction of the superior vena cava and the innominate veins into a collecting chamber or plastic bag, and is pumped from there to the bifurcation of the aorta by way of a cannula in a femoral artery (Fig. 1). The pump is pneumatically cycled with ball-valves at each end of the pumping element.†

An open collection system with weir flow-meter was used for the first eleven procedures. A closed plastic bag was substituted for the open chamber in the last four experiments. Flows were calculated from time collections of 100 c.c. of blood in the chamber or 100 grams in the bag. The closed system was air-free and tightly distended with 800 to 1,100 c.c. of freshly drawn, heparinized, homologous donor blood before the bypass was started. Temperature was controlled by warming a loop of the arterial line in a water bath except in the first two experiments.

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**Of the Brunswick Manufacturing Co., Quincy, Mass.

†Built by the Brunswick Manufacturing Co., Quincy, Mass.

Polyethylene catheters were placed with their tips in the intrathoracic inferior vena cava, at the arch of the aorta, and in the aorta at diaphragm level. Samples drawn from these locations and from the bypass line were analyzed for oxygen content and saturation, carbon dioxide content, pH, hematocrit, electrolytes, and clotting factors. Blood buffer base and pCO_2 were derived from the Singer-Hastings nomogram.[1] Control arteriovenous differences were calculated from the formula:

$$\text{AV difference} = A - \frac{1}{3}(S + 2I)$$

where A = arterial oxygen content
S = superior vena caval oxygen content
I = inferior vena caval oxygen content

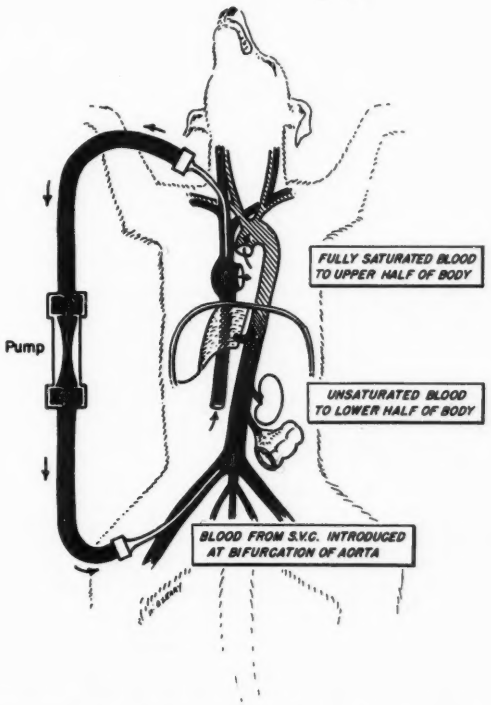

Fig. 1.—Unsaturated blood is withdrawn from the superior vena cava and pumped into the aorta at the bifurcation to perfuse the legs, back, and lower abdomen. The oxygen saturation of blood supplying the heart, brain, and upper abdomen is unaffected.

When the bypass flow was greater than 30 c.c./Kg./min. the pulmonary arteriovenous difference was calculated from the upper aortic and the inferior caval oxygen contents. In one experiment, the cardiac output during perfusion was estimated from the change in pulmonary arteriovenous oxygen difference, assuming that the oxygen consumption was constant. The presence of hemolysis

TABLE I. EFFECTS OF VENO-ARTERIAL SHUNTING: 22 TEST PERIODS IN 9 ANIMALS

CONTROL VALUES	DOG NO.	BLOOD PRESSURE SYST. / DIAST. (MM. Hg)	BODY TEMPERATURE (DEGREES C.)	VOLUME OF VENO-ARTERIAL SHUNT (C.C./Kg./MIN.)	OXYGEN SATURATION (%) AORTA ARCH	AORTA DIAPHRAGM LEVEL	BYPASS LINE	IVC	PULMONARY ARTERIO-VENOUS O_2 DIFFERENCE (C.C./L.)	ARTERIAL pCO_2 (MM. Hg)	BUFFER BASE (mEq./L.)
Mean		178/132	37.7		98	99	85	72	37	36	45
Upper limit		240/165	38.3		103	104	92	78	46	45	50
Lower limit		130/100	37.0		89	94	79	62	23	27	41
	2	130/100	37.8	25	102	107	86	86	36	--	--
	4	160/145	37.1	26	93	92	66	50	--	--	--
	2	130/100	36.2	28	97	102	87	79	44	--	40
	3	200/155	37.6	33	93	101	78	70	62	--	--
	14	180/150	36.0	34	--	110	91	72	76	34	40
	13	210/160	37.8	34	--	88	76	52	73	57	38
	14	240/150	38.0	36	--	86	74	59	57	32	40
	14	220/120	37.8	36	--	110	66	51	91	33	36
	1	140/95	37.4	38	99	100	73	--	--	26	52
	3	200/160	38.1	42	99	98	88	65	73	--	--
	4	175/145	35.0	43	94	99	61	48	113	--	--
	15	165/120	36.4	43	--	107	57	46	106	34	33
	5	150/115	37.0	45	98	93	83	67	55	32	47
	5	160/130	36.2	45	94	97	85	65	75	44	45
	15	150/100	37.4	46	--	95	69	58	55	50	31
	15	160/100	37.6	46	--	99	75	48	67	34	30
	3	205/170	38.0	53	100	94	77	58	86	--	--
	4	165/140	34.6	54	101	98	57	50	114	30	46
	15	170/120	38.0	57	--	110	59	42	77	28	30
	3	200/165	38.2	63	97	77	74	54	93	18	47
	7	145/110	37.6	64	93	102	52	37	124	33	45
	7	170/140	37.7	71	101	100	55	40	126	37	45

was detected by inspection of the plasma in hematocrit tubes after centrifuging. Aortic pressure was constantly recorded on direct-writing paper using a P23Db strain gauge and Sanborn 67 series Poly-Viso.

The last 9 bypasses were started at a rate of 100 to 200 c.c. per minute for 10 minutes before full flow was attempted. When a pronounced drop in arterial pressure occurred during the initial blood exchange, bypass was stopped and the animal allowed to recover for 30 to 45 minutes before bypass was resumed at full flow.

Indicator dilution curves were recorded on direct-writing paper using indigo carmine dye and a Colson constant-flow densimeter system. Dye was injected into the inferior vena cava and the concentration recorded in blood from the aortic arch.

Blood for priming the system and replacement of loss was drawn from donor animals by exsanguination under light Pentothal anesthesia. The heparin concentration in the collection bottles ranged from 23 to 51 mg. per liter. Donor and subject animals were cross-matched in the last three experiments. The subject animal was lightly anesthetized with Pentothal and maintained with 25 to 75 mg. doses of Nembutal as required. The trachea was intubated and oxygen run in through a catheter at a flow of 2 L. per minute. The animals were allowed to breathe spontaneously. The total oxygen content of arterial blood is expressed as a percentage of hemoglobin capacity without correction for dissolved oxygen.

Each subject received 2 mg. per kilogram of body weight of heparin initially. Heparinization was maintained with doses of 50 to 100 mg. every 3 hours, except in the last experiment in which a continuous infusion was given.

Cineangiography was performed in 2 animals using a Phillips image intensifier and 16 mm. movie film. Ninety per cent Hypaque, at a concentration of 0.66 c.c. per kilogram, was injected into the femoral artery cannula during maximal bypass.

RESULTS

Fifteen animals were studied. In 6 animals, hypotension and normovolemic shock occurred a few minutes after the onset of veno-arterial pumping. Five of these were excluded from the analysis. The sixth animal recovered rapidly and, as final control samples were similar to those taken before the onset of shock, was included in the study. In 5 dogs, long-term perfusion was undertaken.

Effects of Veno-Arterial Shunting at Various Rates.—Twenty-two test periods in 10 animals were found suitable for evaluation. Six further test periods were excluded because of respiratory depression, uncontrolled cooling, or a diastolic blood pressure below 90 mm. Hg. The volume of the veno-arterial shunt ranged from 25 to 71 c.c./Kg./min. (Table I).

There was no significant change in mean arterial blood pressure during perfusion. However, the pulse pressure was considerably reduced with elevation of the diastolic pressure (Fig. 2).

The oxygen saturation of blood in the aorta at the arch and at the diaphragm was unchanged by veno-arterial pumping, except in one test period at 63 c.c./Kg./min. in which the oxygen saturation at the diaphragm fell to the level found in the shunt line.

The oxygen saturation of blood from the bypass line ranged from 79 to 92 per cent in the control samples, while the inferior vena caval saturation was between 62 and 78 per cent. With increasing veno-arterial pumping, both bypass line and inferior caval oxygen saturations tended to fall. In the 6 test periods

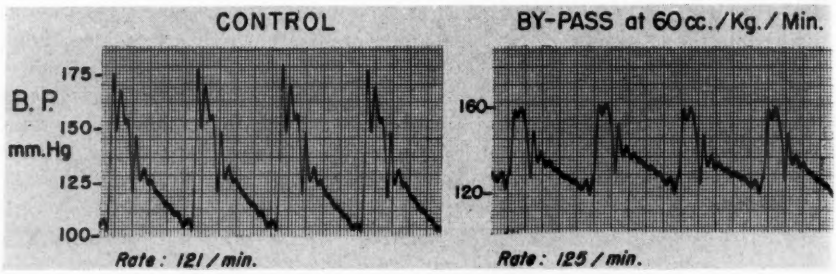

Fig. 2.—The effect of veno-arterial pumping on the aortic pressure pulse. During perfusion the pulse pressure is diminished and the diastolic pressure is elevated.

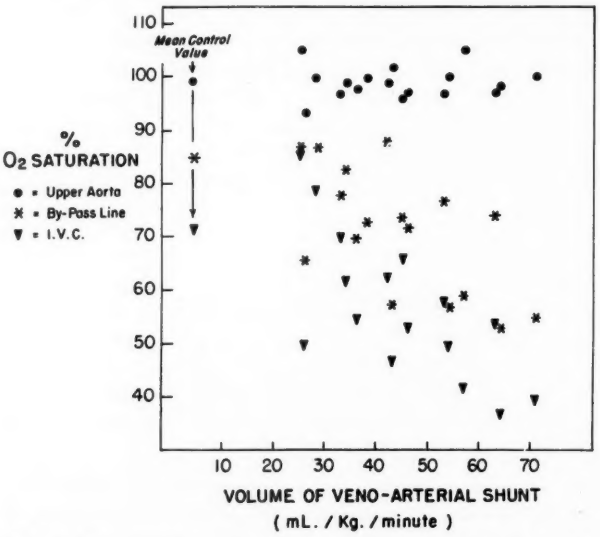

Fig. 3.—The oxygen saturation in the upper aorta is unaffected by veno-arterial shunting. The saturation in the bypass line and in the inferior vena cava falls progressively with increasing perfusion.

with a bypass flow greater than 50 c.c./Kg./min., the bypass line saturation averaged 62 per cent and the inferior caval saturation 47 per cent (Fig. 3).

The pulmonary arteriovenous oxygen difference increased progressively with the volume of the veno-arterial shunt above values of 30 c.c./Kg./min.

(Fig. 4). In Dog 3 the cardiac output was estimated during perfusion. The sum of cardiac output and bypass flow was between 101 and 108 c.c./Kg./min. in four test periods. The average cardiac output determined by the dye dilution method in 5 control periods was 105 c.c./Kg./min.

The arterial pCO_2 showed little change. The buffer base was normal in the control period and was not consistently affected by veno-arterial pumping. The low values in Dog 15 were attributed to hemorrhage (Table I).

Cineangiography with bypass flows of 58 and 60 c.c./Kg./min. showed that the shunt blood filled the aorta and its branches to just below the level of the diaphragm. The upper level of the opacified blood fluctuated with the heartbeat.

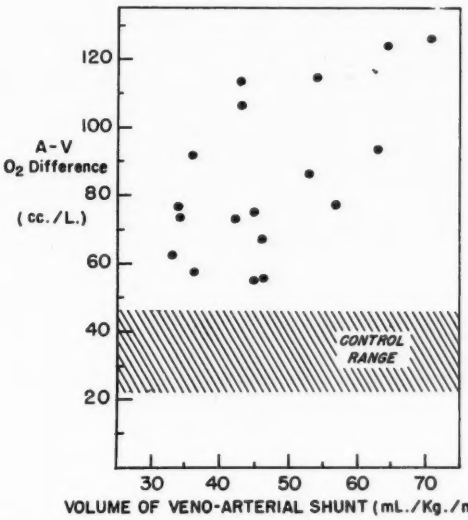

Fig. 4.—The arteriovenous oxygen difference across the lungs increases with the bypass flow, indicating a progressive reduction in the output of the heart.

The renal and hepatic arteries were filled with dye blood only during ventricular diastole (Fig. 5).

Prolonged Veno-Arterial Shunting.—Long-term studies were undertaken in 5 animals (Table II). In 4, the procedure was terminated by the death of the animal. Dog 14, however, survived after 52 hours' perfusion.

TABLE II

DOG NO.	VOLUME OF VENO-ARTERIAL SHUNT (C.C./KG./MIN.)	DURATION OF SATISFACTORY PERFUSION (HOURS)	TOTAL DURATION OF PERFUSION (HOURS)	CAUSE OF TERMINATION OF PROCEDURE	MEAN DOSE OF HEPARIN (MG./KG./HR.)
2	25	14	19	Uncontrolled cooling	0.58
3	42	16	17	Aspiration of vomit	0.68
5	44	13	24	Probable incompatible transfusion	0.81
14	36	52	53	Occlusion of pump by clot	1.18
15	45	35	39	Continued blood loss	1.60

In the first experiment, uncontrolled fall in body temperature to 32° C. was associated with a fall in blood pressure and blood oxygen content, and eventual death after 19 hours. In subsequent animals, normal body temperatures were maintained. In Dog 5, a fall in blood pressure and oxygen saturation after 13 hours followed transfusion from a new donor. The next experiment was satisfactory for 52 hours when the pump became occluded by a thrombus (Fig. 6). The animal recovered rapidly from the anesthetic and no untoward aftereffects were noted. The final dog (No. 15) was difficult to manage because of continued hemorrhage from operation sites.

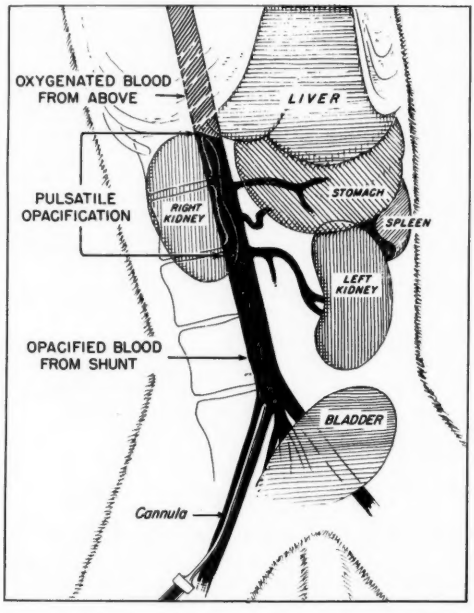

Fig. 5.—This diagram is constructed from a cineangiographic study in which the bypass flow was opacified. At a shunt flow of 60 c.c./Kg./min. unsaturated blood reaches the level of the diaphragm. Ventricular systole, however, fills the renal and hepatic arteries with fully saturated blood.

During these prolonged experiments, a normal urine volume was maintained, and the blood nonprotein nitrogen rose only from an average of 22 to 31 mg. per 100 c.c. The electrolyte pattern of the plasma was little disturbed.

Effect of Prolonged Pumping on the Coagulation Mechanism.—In the first three experiments the changes were uniform. The fibrinogen content was little affected, and the measurements of prothrombin, proconvertin, and Stuart factor, together with the Quick "prothrombin" time, showed moderate prolongation (Fig. 7). The platelet count fell in the first hour to approximately 100,000 per cubic millimeter, but showed no subsequent change. In the fourth dog, there were similar changes until the forty-eighth hour when the platelet count fell to 40,000 per cubic millimeter and the fibrinogen to 227 mg. per 100 c.c. Clotting in the apparatus was noted a few hours later. In the fifth animal, a larger

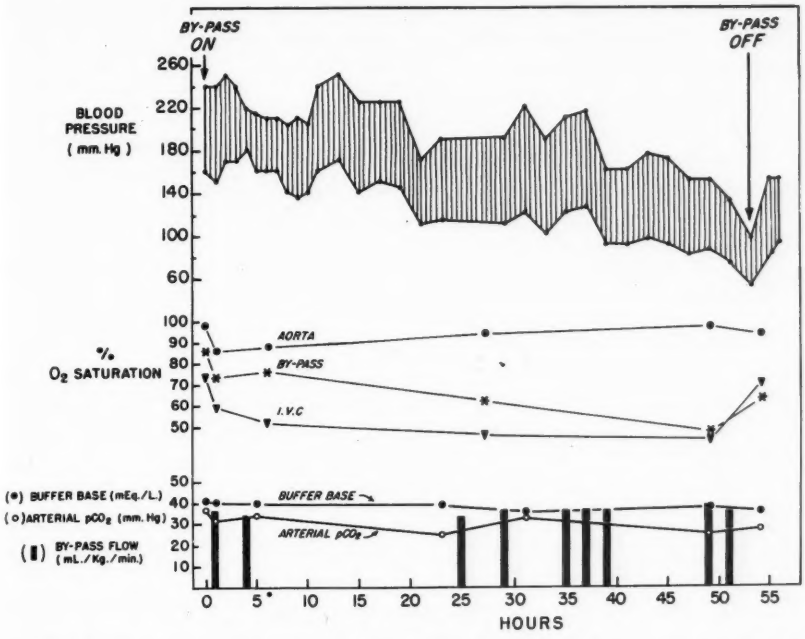

Fig. 6.—Dog 14 was perfused at 36 c.c./Kg./min. for 52 hours with survival. The bypass was terminated when thrombus occluded the pump. A fall in blood pressure at this time was due to temporary loss of blood into the collection bag.

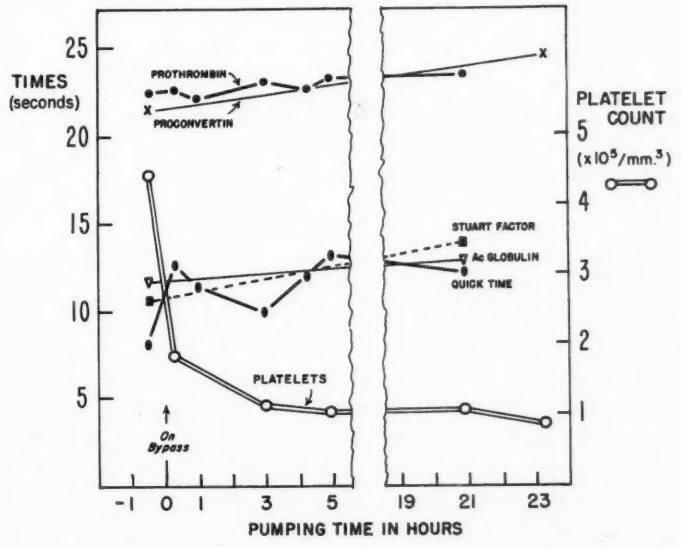

Fig. 7.—Changes in coagulation factors during prolonged veno-arterial shunting in Dog 5.

over-all dose of heparin was given by continuous infusion and hemorrhage became a problem. The changes in coagulation factors were, however, similar to those in the previous experiments.

Hemolysis.—No consistent changes in hematocrit, hemoglobin, or red cell count were found. Hemolysis was noted in the first blood samples after the onset of perfusion in 5 of the 15 animals. In 3 of these 5, the apparatus had been primed with mixed blood from 2 donor dogs. Late hemolysis was found only in 3 animals in which blood from a second donor dog was used for transfusion after several hours.

DISCUSSION

At low rates of veno-arterial pumping, blood is drawn from the superior vena cava only, and is 70 to 80 per cent saturated with oxygen. This blood supplies the lower extremities. At shunt flows greater than 30 c.c./Kg./min., the rate of superior vena cava return is exceeded, and the shunt line draws increasingly from the inferior vena cava. At flows of 60 c.c./Kg./min., the shunt mixture is composed of roughly equal parts of superior and inferior caval blood. Part of the coronary sinus flow is presumably also drawn off. In consequence, the lower part of the body is perfused by progressively larger volumes of more reduced blood.

Most of this oxygen-poor blood goes to supply the muscle mass of the lower extremities and back. At shunt flows of 58 and 60 c.c./Kg./min., cineangiograms show the dyed interface to oscillate at mid-abdominal level in the aorta (see Fig. 5). The shunted blood fills the renal arteries and reaches as high as the celiac axis during diastole, but is forced back past the renal vessels by ventricular systole. The splanchnic bed is therefore perfused in large part by freshly oxygenated blood even at the highest veno-arterial flows attained.

Progressive widening of the arteriovenous oxygen difference across the lung provides evidence that cardiac output declines as the veno-arterial shunt increases (see Fig. 4), and suggests that total body perfusion remains constant. Experiment No. 3, in which the sum of bypass flow and cardiac output is similar to the control cardiac output determined by the dye dilution method, supports this conclusion.

In addition to saturation of arterial blood, oxygen availability is determined by the rate of blood flow and of oxygen utilization. In these experiments, total tissue flow is maintained, and arterial blood supplying coronary, cerebral, and most of the splanchnic beds is fully oxygenated at shunt volumes up to 50 c.c./Kg./min. The low saturation of the final venous mixture obscures the fact that contributions to this mixture from the vital organs cannot be poor in oxygen.

If total body perfusion is unchanged, the volume output of the heart is reduced quantitatively by the volume of the veno-arterial shunt. This reduction is reflected in the narrowed aortic pressure pulse (see Fig. 2). At the same time, the higher diastolic pressure should tend to increase the myocardial blood flow.

SUMMARY

1. A system for diverting part of the venous return from the venae cavae and pumping it into the aorta at the bifurcation has been devised.

2. The maximum rate of pumping achieved was 71 c.c./Kg./min., and the longest duration of satisfactory perfusion was 52 hours.

3. Total tissue perfusion is not changed by pumping, so that the volume output of the heart is reduced by the amount of the shunt.

4. The oxygen saturation of shunted blood falls progressively when the shunt is increased beyond 30 c.c./Kg./min. At flows from 50 to 71 c.c./Kg./min., the oxygen saturation of shunted blood averages 62 per cent.

5. Oxygenation of the splanchnic bed is normal at shunt flows less than 50 c.c./Kg./min.

6. Veno-arterial pumping for 52 hours did not cause major irreversible changes in blood, renal function, or acid-base balance.

Our thanks are due to Dr. Herbert S. Sise and his colleagues of the Anticoagulant Laboratory, Dr. William C. Moloney and Dr. Jane Desforges of the Hematology Laboratory, Dr. C. Gardner Child III for the use of the apparatus for cineangiography, and Dr. John J. Byrne for the use of the facilities of the Boston University 3rd Surgical Research Laboratory at the Boston City Hospital; also, to Miss JoAnn Harris for surgical assistance, Miss Faye Baer for the blood gas analysis, and Miss Violet Peters for her help in preparing the manuscript.

ADDENDUM

This technique has recently been used to support the circulation in a patient with intractable congestive heart failure. Veno-arterial pumping was performed for 26 hours at a rate of 1 L. per minute. There was no evident change in the patient's condition as a result of the procedure, and he died 2 days later. Autopsy showed no lesions attributable to the apparatus. A striking feature was the plasma hemoglobin concentration, which was only 5 mg. per cent after 16 hours' pumping. Evidence from the oxygen saturations suggests that the pumped blood was diverted to areas of low vascular resistance in the lower part of the body. A larger extracorporeal flow might have been more effective. Nevertheless, this experience demonstrates that veno-arterial pumping is a practical procedure for use in man.

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THE PHONOCARDIOGRAM AS A METHOD FOR EVALUATING MITRAL STENOSIS: CORRELATION WITH OPERATIVE FINDINGS

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IN RECENT YEARS, phonocardiography has been utilized to estimate the severity of mitral stenosis. The interval between the onset of electrical activation of the ventricle and the closure of the mitral valve (Q-1 interval), measured by means of simultaneous phonocardiogram and electrocardiogram, has been found usually to be prolonged in mitral stenosis.[1-4] Similarly, a shortened interval between the second heart sound and the characteristic opening snap of mitral stenosis (2-OS interval) has been thought to indicate the severity of the stenosis.[5] In mitral stenosis, these variations have been attributed to the pressure gradient across the mitral valve.[6, 7] It appears likely that, in predominant mitral stenosis, a delayed first sound results from elevation of left atrial pressure. Late equilibration of atrial and ventricular pressures during ventricular systole produces delayed closure of the mitral valve. Similarly, early equilibration of these pressures during the stage of isometric ventricular relaxation, because of elevated atrial pressure, would produce an early opening snap.

The diagnostic implications of prolongation of the Q-1 interval and of the time relationship between the second sound and the opening snap have led to investigations of the usefulness of these determinations in evaluating patients for mitral valve surgery. Bayer and associates[5] constructed a curve from simultaneous mean pulmonary capillary pressure and 2-OS intervals in 100 patients which roughly corresponded to the terminal slope of the ventricular isometric relaxation curve and indicated that the left atrial pressure could be thus estimated. Wells[6] examined a series of 30 patients and correlated the size of the mitral orifice at surgery with the Q-1 and 2-OS intervals and the difference between these intervals which he termed the Q-1 minus 2-OS value. Because of variations in these intervals with heart rate, he first analyzed a group with auricular fibrillation and found an approximate linear relationship between cycle lengths of 0.6 and 1.2 seconds for both the Q-1 and the 2-OS

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intervals. He thus was able to find "corrected" values for a cycle length of 0.8 second. Apparently using multiple observations in cases of sinus rhythm, he obtained corrected values in these cases also. The best correlation was thought to exist between mitral valve size and the "corrected" Q-1 minus 2-OS value. He thought that any delay in the individual intervals resulting from rigidity of the valve cusps would be offset by using both intervals for estimate.

The present study was undertaken to evaluate independently the usefulness of these determinations in the selection of patients for surgery.

METHOD

Forty-seven patients were studied by means of preoperative phonocardiograms. Estimations of valve size were made in each instance by the same surgeon* by means of insertion of the finger tip into the valve orifice at time of surgery. Intervals were measured from logarithmic phonocardiographic tracings taken at the apex and simultaneous Lead II electrocardiograms using a Sanborn Twin-Beam Cardiette. The Q-1 interval was measured from the onset of the QRS complex to the first major vibration of the first heart sound. The 2-OS interval was measured from the beginning of the second heart sound to the onset of the opening snap. Opening snaps were recorded in all but 3 patients. No patients had bundle branch block or discernible intraventricular conduction delay which might prolong the Q-1 interval.[3] One patient had 2:1 atrioventricular block.

Fifteen of the 47 patients had auricular fibrillation. Of these, 13 had detectable opening snaps. Assuming a linear relationship between the time intervals and the change in preceding cycle length between cycle lengths of 0.6 to 1.2 seconds, linear regression lines were calculated. Statistically significant values for the slope of these lines were obtained only when variation in cycle length exceeded 0.3 second. Such a range is required to produce measurable variations in the time intervals. The "corrected" Q-1 and 2-OS values could be calculated by substitution in the individual cases.

The mean regression coefficient with standard error was -0.048 ± 0.004 for the Q-1 intervals and 0.054 ± 0.004 for the 2-OS intervals. These figures for linear regression are essentially in agreement with Wells's observations of a decrease in Q-1 of 0.03 second and an increase in 2-OS of 0.03 second with change in cycle length from 0.6 to 1.2 seconds.

In our experience, regression lines could not be calculated in patients with normal sinus rhythm because of the narrow variations in cycle length. In those cases, after obtaining the means of multiple Q-1 and 2-OS intervals, correction of the intervals to a cycle length of 0.8 second was made by assuming a decrease of 0.005 second in Q-1 time and an increase of 0.005 second in 2-OS time for each increase of 0.1 second in cycle length. The validity of these corrections may be open to question.

*Dr. Will Sealy, Professor of Thoracic Surgery.

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Postoperative studies were carried out on 17 patients, 7 of whom had auricular fibrillation.

RESULTS

For general assessment, the patients have been grouped into four categories based on valve sizes of less than 0.5 cm.^{2}, 0.5 to 1.0 cm.^{2}, 1.0 to 1.5 cm.^{2}, and greater than 1.5 cm.^{2}.

Estimates of insufficiency and calcification were also made at operation. Scattergraphs (Figs. 1, 2, 3) show the correlation between the "corrected" Q-1 intervals, 2-OS intervals, and Q-1 minus 2-OS values and the mitral valve size at surgery. The Q-1 interval failed to disclose satisfactory predictive correlation with valve size. With five exceptions, the Q-1 interval was 0.07 second or greater in each of the valve size groups, and was 0.08 second or greater in 8 of 13 patients whose valve size was greater than 1.0 cm.^{2}.

However, reasonably good correlation is apparent for both the 2-OS interval and the Q-1 minus 2-OS value. Twenty-nine (85 per cent) of 34 patients, whose valve areas measured 1.0 cm.^{2} or less, had a 2-OS interval of 0.09 second or less in comparison to 2 of 10 patients whose valve size exceeded 1.0 cm.^{2}. All 11 patients whose valve area was less than 0.5 cm.^{2} exhibited a 2-OS interval of 0.09 second or less, as well as 18 of 23 patients whose valve size ranged from 0.5 to 1.0 cm.^{2}. Eight of 10 patients in whom the estimated valve size was greater than 1.0 cm.^{2} showed a 2-OS interval of 0.095 second or greater. Thirty (90 per cent) of 34 patients, whose valve area measured 1.0 cm.^{2} or less, exhibited Q-1 minus 2-OS values of –1.0 or greater, as compared to 3 of 10 patients whose valve size exceeded 1.0 cm.^{2}. Utilizing values of 0.09 second for 2-OS interval and –1.0 for Q-1 minus 2-OS difference, seven errors in prediction of valve size are noted for each of these indices employed alone. The estimated valve size at operation was smaller in 5 patients and greater in 2 patients than predicted from the 2-OS interval. Valve size was smaller in 4 patients and larger in 3 patients than predicted from the Q-1 minus 2-OS value. Because of overlap, these 14 prediction errors involve only 9 patients. However, when the values for both indices were satisfied as criteria of valve size, prediction errors remained in only 5 of the group of 44 patients; valve size was overestimated in 3 and underestimated in 2 patients.

The 17 postoperative studies showed changes in Q-1 and 2-OS intervals similar to those described by others.[3-6] The individual pre- and postoperative values in these cases are summarized in Table I. Five patients showed shortening of the Q-1 time compared to preoperative values while an additional 6 showed slight increases (Fig. 4). In 14 patients there was lengthening of the 2-OS interval (Fig. 5). Thirteen patients exhibited decreases in Q-1 minus 2-OS values, the change being greater in those patients presenting more severe mitral stenosis (Fig. 6). In 1 of the remaining 4 patients (Patient L. H.), pure mitral insufficiency was present and valvulotomy was not performed.

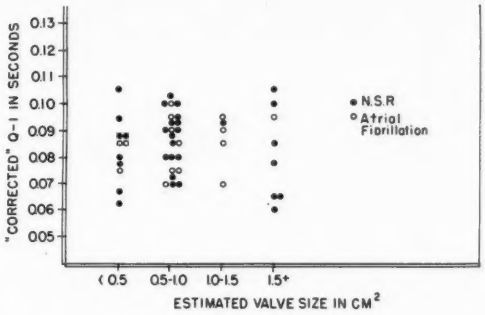

Fig. 1.—Scattergraph demonstrating relationship between estimated valve size and "corrected" Q-1.

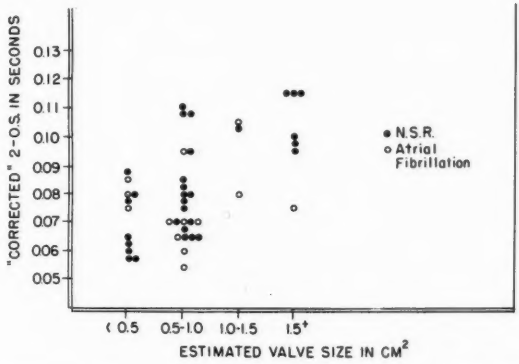

Fig. 2.—Scattergraph demonstrating correlation between valve size and "corrected" 2-OS.

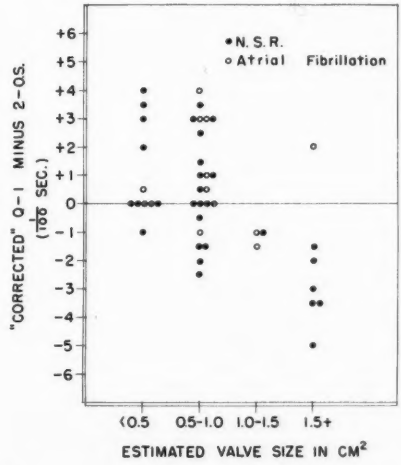

Fig. 3.—Scattergraph demonstrating correlation between estimated valve size and "corrected" Q-1 minus 2-OS.

TABLE I. COMPARISON OF PRE- AND POSTOPERATIVE VALUES FOR "CORRECTED" Q-1, 2-OS, Q-1 MINUS 2-OS INTERVALS AND VALVE SIZE

	BEFORE VALVULOTOMY					AFTER VALVULOTOMY				
PATIENT	RHYTHM	Q-1 IN SECONDS	2-OS IN SECONDS	Q 1 MINUS 2-OS	ESTIMATED VALVE AREA IN CM.^{2}	RHYTHM	Q-1 IN SECONDS	2-OS IN SECONDS	Q-1 MINUS 2-OS	ESTIMATED VALVE AREA IN CM.^{2}
L. B.	NSR	.088	.058	+3.0	0 5	NSR	.088	.068	+2.0	2.0
T. F.	Aur. Fib.	.085	.08	+0.5	0.5	Aur. Fib.	.085	.11	−2.5	3.5
H. C.	NSR	.088	.088	0.0	0.5	NSR	.09	.10	−1.0	1.0-1.5
F. B.	NSR	.08	.08	0.0	0.5	NSR	.083	.093	−1.0	2.5
D. D.	2:1 AVB	.10	.065	+3.5	0.5-1.0	2:1 AVB	.10	.08	+2.0	3.0
V. P.	Aur. Fib.	.10	.07	+3.0	0 5-1.0	Aur. Fib.	.075	.085	−1.0	3.5
V. H.	Aur. Fib.	.09	.06	+3.0	0.5-1.0	Aur. Fib.	.075	.11	−3.5	2.5
G. E.	NSR	.093	.078	+1.5	0.5-1.0	NSR	.085	.085	0.0	3.0
P. O'D.	NSR	.085	.075	+1.0	0.5-1.0	NSR	.093	.108	−1.5	3.0
L. P.	Aur. Fib.	.075	.065	+1.0	0.5-1.0	Aur. Fib.	.075	.065	+1.0	3.0
J. H.	NSR	.093	.083	+1.0	0.5-1.0	NSR	.10	.11	−1.0	3.0
C. B.	Aur. Fib.	.075	.07	+0.5	0.5-1.0	Aur. Fib.	.07	.085	−1.5	3.0
W. W.	NSR	.08	.08	0.0	0.5-1.0	NSR	.078	.093	−1.5	2.5
M. J.	NSR	.07	.085	−1.5	0.5-1.0	NSR	.085	.09	−0.5	3.5
J. S.	Aur. Fib.	.09	.105	−1.5	1.0-1.5	Aur. Fib.	.10	.12	−2.0	2.5-3.0
E. H.	Aur. Fib.	.095	.075	+2.0	1.5	Aur. Fib.	.095	.065	+3.0	3.0
L. H.	NSR	.065	.10	−3.5	4.0	NSR	.065	.10	−3.5	4.0

NSR—Normal sinus rhythm; Aur. Fib.—Auricular fibrillation; AVB—Atrioventicular block.

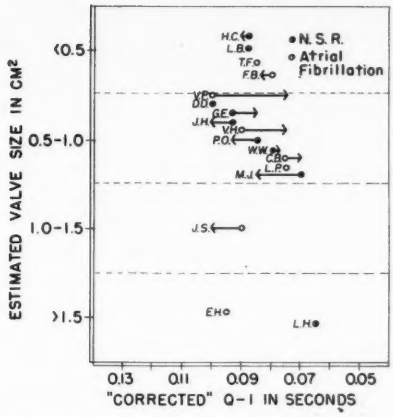

Fig. 4.—Individual changes in Q-1 values after operation (patients grouped according to valve size).

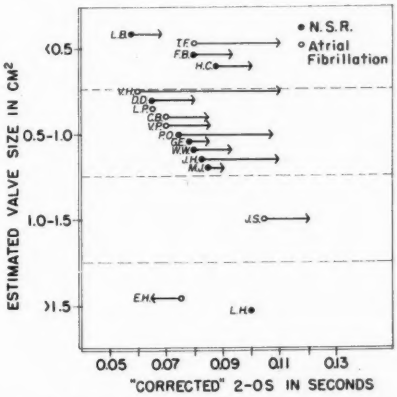

Fig. 5.—Individual changes in 2-OS values after operation (patients grouped according to valve size).

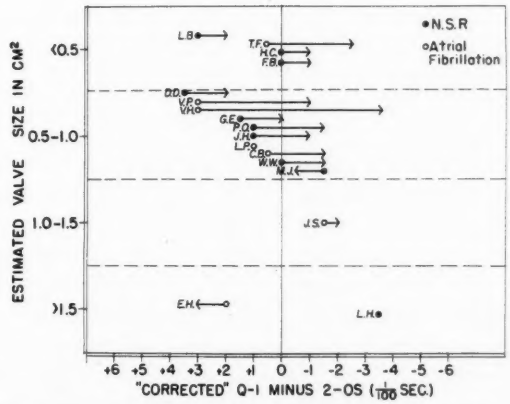

Fig. 6.—Individual changes in Q-1 minus 2-OS values after operation (patients grouped according to valve size).

DISCUSSION

The duration of the Q-1 interval failed to reveal appreciable correlation with valve size in this series. These observations are not in agreement with conclusions of others. Kelly[3] felt that, in the absence of bundle branch block, prolongation of the Q-1 interval was found only with mitral stenosis and that a Q-1 interval of 0.07 second or more indicated significant stenosis. Such a Q-1 interval range would encompass all save 5 patients in our entire group whose valve sizes varied from less than 0.5 cm.^{2} to greater than 1.5 cm.^{2}. Moreover, 8 of 13 patients who presented valve areas greater than 1.0 cm.^{2} had Q-1 intervals of 0.08 second or longer. Reasonably good correlation was found between valve size and the 2-OS interval. A short 2-OS interval, noted by most investigators in severe mitral stenosis, is thought to reflect elevated left atrial pressure.[3, 5, 6] An analysis of the scattergraph of our observations (Fig. 2) revealed that 29 (85 per cent) of 34 patients, whose valve areas measured 1.0 cm.^{2} or less, had 2-OS intervals of 0.09 second or less in comparison to 2 of 10 patients whose valve size exceeded 1.0 cm.^{2}. Each of the 11 patients whose valve area was less than 0.5 cm.^{2} had a 2-OS interval of less than 0.09 second. Steinzeig and associates[4] found less consistent shortening of the 2-OS interval but no comparisons were made with less severe cases or those without significant stenosis. This study demonstrates satisfactory correlation of mitral valve size with the Q-1 minus 2-OS value as noted by Wells.[6] Thirty (90 per cent) of 34 patients, whose valve areas measured 1.0 cm.^{2} or less, had a "corrected" Q-1 minus 2-OS value of –1.0 or greater as compared to 3 of 10 patients whose valve size exceeded 1.0 cm.^{2}. Six of 7 patients, whose valve size was greater than 1.5 cm.^{2}, had values more negative than –1.0. Wells[6] suggested that the use of the Q-1 minus 2-OS value would offset any prolongation of the Q-1 or 2-OS intervals caused by rigidity of the valve cusps. However, in our series, 6 patients with heavy valvular calcification showed no consistent lengthening of either interval over noncalcified cases with the same valve areas. Wells subsequently has reported on a group of 100 patients who had been studied preoperatively with phonocardiograms. In this group the mitral pressure gradient, determined at operation, was found to be high in patients who had a "corrected Q-1 minus 2-OS" greater than –1.5, while the pressure gradient was found to be low in patients who had a value less than –1.5.[8] Our determinations and correlation with valve size are in general agreement with those values.

In the group of patients considered, surgical intervention was decided mainly upon clinical grounds, sometimes assisted by right heart catheterization. With increasing use of left heart catheterization, phonocardiography may prove a useful adjunct to clinical evaluation. In patients suspected of having tight mitral stenosis, a "corrected" 2-OS interval of greater than 0.09 second and a "corrected" Q-1 minus 2-OS value more negative than –1.0 should lead to careful re-evaluation and, if necessary, left heart catheterization before surgery is undertaken. On this basis alone, 6 of the 7 patients, whose valve size at operation was found greater than 1.5 cm.^{2}, would now be recommended for left heart catheterization before surgery.

Recognizing significant agreement but with definite limitations, it appears that routine phonocardiography will assist in screening potential candidates for mitral valvulotomy. If the 2-OS interval is less than 0.09 second and the Q-1 minus 2-OS value is greater than –1.0 in the same patient, the likelihood of a fairly tight mitral stenosis is good. These indices, used together as criteria of valve size, possess greater value than either index used alone.

SUMMARY

The value of phonocardiography in selecting candidates for mitral commissurotomy has been assessed in a series of 47 patients submitted to surgery. Comparisons between the "corrected" figures for the Q-1 intervals, 2-OS intervals, and Q-1 minus 2-OS values and the size of mitral orifice found at operation have been made. Appreciable correlation was evident between valve size and both the 2-OS interval and the Q-1 minus 2-OS value. No significant correlation was observed between the Q-1 interval and valve size. Postoperative observations in 17 patients revealed shortening of the Q-1 time in 5 patients, lengthening of the 2-OS interval in 14 patients, and decrease in Q-1 minus 2-OS value in 13 patients, the changes being greater in those patients exhibiting more severe mitral stenosis. When both the 2-OS interval is less than 0.09 second and the Q-1 minus 2-OS value is greater than –1.0 the likelihood of significant mitral stenosis is great. Fewer predictive errors in valve size are made when the values observed in a single patient satisfy both indices rather than either index used alone as a criterion.

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CARDIAC PACEMAKER AND DEFIBRILLATOR WITH VARIABLE OUTPUT

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IT HAS BEEN demonstrated that electrical stimuli may be used to induce myocardial fibrillation, to defibrillate the myocardium, or to control the heart action. The response of the myocardium is determined by the characteristics of the stimulus. Alternating current (60 c.p.s.) of less than 28 volts will cause fibrillation (never defibrillation) and of greater than 85 volts will cause defibrillation (never fibrillation). Similarly, 0.5 amp. (60 c.p.s.) will cause fibrillation (never defibrillation) and 1.5 to 3.8 amp. will cause defibrillation (never fibrillation).[1] Further studies have shown that higher voltages (270) are more effective for defibrillation[2] and that short impulses are less likely to result in myocardial damage.[3] However, the optimum voltage-time relationships have not been precisely established and may vary.

The optimum stimulus for control of heart action has been more accurately determined. A pacemaker pulse may be monophasic or biphasic but should be single with a sharp rise and exponential decay. The optimum duration of pulse is 0.02 to 4 milliseconds. Voltage requirement to initiate systole is usually 2 to 3 volts (less than 20 volts).[4, 5, 6, 7] Repetitive stimulation within physiologic range is desirable (up to 120 per minute).

For the past 4 years we have used a defibrillator and pacemaker of our own design.† This instrument is constructed to allow a large variation in output while being of comparatively simple design and relatively inexpensive.

CHARACTERISTICS OF APPARATUS

The defibrillator produces pulses of up to 300 volts at 60 cycles per second. The range of frequency of the output impulses is 0 to 400 per minute. The duration of each output impulse is continuously variable between 0 per cent and 50 per cent of the interval between the initiation of successive impulses (Fig. 1; Tables I and II). It is therefore possible to set the output to

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†Manufactured for us by Barry Johnson, 85 Gerry Rd., Brookline, Mass.

deliver impulses of any voltage up to 300, any rate up to 400 per minute, and any desired pulse width down to one half of one cycle of the alternating current.

Pacemaker.—The output voltage of the pacemaker is continuously variable between 0 and 20 volts. The output impulses are variable between 0 and 200 per minute. Each impulse is a square wave rising sharply and decaying to 60 per cent of maximum in 3 milliseconds, one such wave being emitted per pulse (Fig. 2).

Fig. 1.

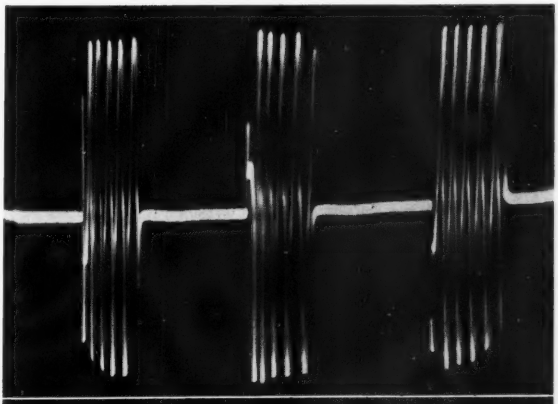

Fig. 2.

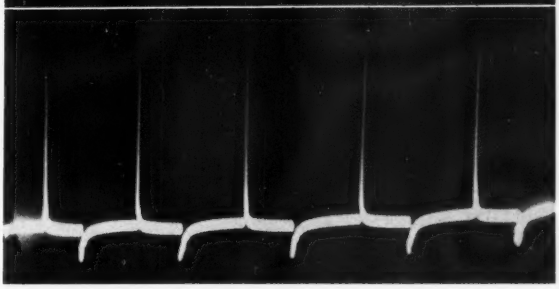

Fig. 1.—Defibrillator output as seen on oscilloscope.
Fig. 2.—Pacemaker output as seen on oscilloscope.

PRINCIPLES OF OPERATION

Defibrillator.—The 60-cycle input current is stepped up to 300 volts by means of a transformer (Fig. 3) and then fed to a variable autotransformer controlling the output voltage. A pair of motor-driven cam-operated switches are connected in series between the two transformers. These switches serve to open and close the circuit at the desired rate, determined by the speed of rotation of the cam that actuates the switches. The rotational velocity of the cams is controlled by a variable speed mechanism, linking the cam-shaft to the drive motor.

TABLE I. DURATION OF DEFIBRILLATOR PULSE*
FRACTION OF MINUTE

PULSE RATE DIAL SETTING	PULSE WIDTH DIAL SETTING									
	1	2	3	4	5	6	7	8	9	10
(1)†	0.05	0.10	0.15	0.20	0.25	0.30	0.35	0.40	0.45	0.50
0.5 (50)	0.001	0.002	0.003	0.004	0.005	0.006	0.007	0.008	0.009	0.010
1 (100)	0.0005	0.0010	0.0015	0.0020	0.0025	0.0030	0.0035	0.0040	0.0045	0.0050
1.5 (150)	0.00032	0.00064	0.00096	0.00128	0.00160	0.00192	0.00224	0.00256	0.0288	0.00320
2 (200)	0.00025	0.0005	0.00075	0.0010	0.00125	0.00150	0.00175	0.00200	0.00225	0.00250
3 (300)	0.000165	0.00033	0.000495	0.000660	0.000825	0.000990	0.001155	0.001320	0.001485	0.001650
4 (400)	0.000125	0.000250	0.000375	0.000500	0.000625	0.000750	0.000875	0.001000	0.001125	0.001250

*Length of defibrillator output pulse at selected settings of *Pulse Width* and *Pulse Rate* dials. Any desired pulse width within this range may be obtained by setting the dials appropriately.
†( ) = pulses per minute.

TABLE II. DURATION OF DEFIBRILLATOR PULSE*
NUMBER OF CYCLES (60 C.P.S.)

PULSE RATE DIAL SETTING	PULSE WIDTH DIAL SETTING									
	1	2	3	4	5	6	7	8	9	10
(1)†	180	360	540	720	900	1080	1260	1440	1620	1800
0.5 (50)	3.6	7.2	10.8	14.4	18.0	21.6	25.2	28.8	32.4	36
1 (100)	1.8	3.6	5.4	7.2	9.0	10.8	12.6	14.4	16.2	18.0
1.5 (150)	1.2	2.4	3.6	4.8	6.0	7.2	8.4	9.6	10.8	12.0
2 (200)	0.9	1.8	2.7	3.6	4.5	5.4	6.3	7.2	8.1	9.0
3 (300)	0.6	1.2	1.8	2.4	3.0	3.6	4.2	4.8	5.4	6.0
4 (400)	0.45	0.9	1.35	1.80	2.25	2.70	3.15	3.60	4.05	4.50

*Length of defibrillator output pulse in cycles at selected settings of *Pulse Width* and *Pulse Rate* dials.
†( ) = pulses per minute.

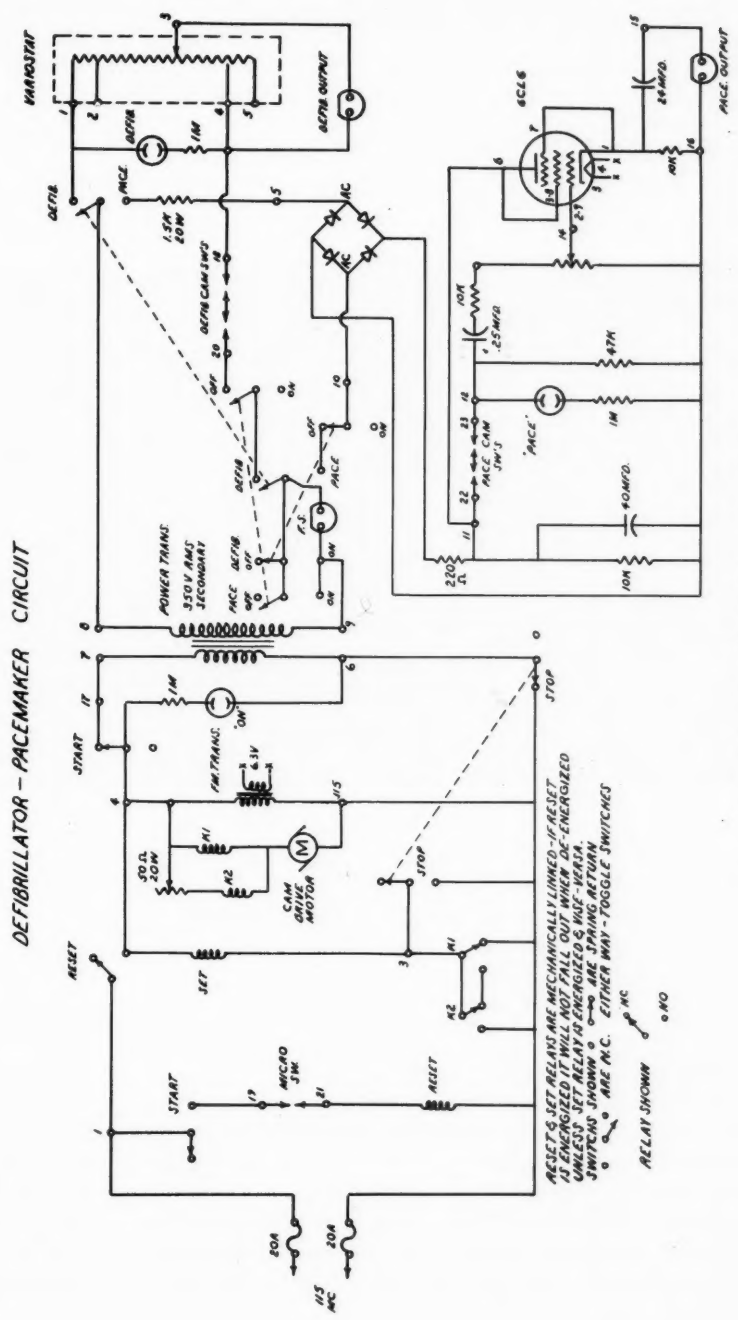

Fig. 3.—Wiring diagram of defibrillator-pacemaker.

The duration of the output impulse is controlled by rotating one of the switches with respect to the other on the common cam-shaft. When the switches are actuated simultaneously, the output is maximum (50 per cent of interval between onset of successive impulses). When one lags the other, the impulse width is diminished as the lag increases, to the point where one switch opens at the same time the other closes, making the impulse width 0.

Pacemaker.—The source of the pacemaker output impulse is a DC power supply. The DC voltage is switched on and off by a single cam-actuated switch.

Fig. 4.—Control panel of unit.

The rotation of this cam is controlled by a variable speed mechanism linking the cam-shaft and the drive motor. The resulting output impulse is a sharp square wave of constant duration which is fed into a differentiating circuit in order to obtain pulses that rise sharply and decay exponentially within 3 milliseconds.

OPERATION

Defibrillator.—The voltage is varied by the front panel control labeled *Defib. Pulse Ampl.* (defibrillator pulse amplitude) (Fig. 4), which is calibrated from 0 to 300. The pulse repetition rate is varied by the control labeled *Defibrillator Pulse Rate,* calibrated from 0 to 4, and indicating rate in hundreds of pulses per minute (0-400). The length of the output impulse is determined by the setting of the *Defibrillator Pulse Width* control. This control is calibrated from 0 to 10 which indicates 0 to 50 per cent of the pulse interval as the duration of the output impulse. The defibrillator electrodes are attached at the

Defib. Output (defibrillator output) connector. The defibrillator *On-Off* switch, which has a spring return to the *Off* position, may be used to energize the defibrillator circuit (rather than using the foot switch). The *Defib. Pacemkr.* (defibrillator pacemaker) selector switch must be in the "defibrillator" position. The *Defib.* (defibrillator) lamp flashes synchronously with each defibrillator impulse and indicates the rate and duration of each impulse.

Pacemaker.—Output voltage is controlled by the *Pacemkr. Pulse Ampl.* (pacemaker pulse amplitude), calibrated from 0 to 20 (volts). The *Pacemkr. Pulse Rate* (pacemaker pulse rate) controls the rate of pacemaker pulses and is calibrated from 0 to 2, indicating the pulse repetition rate in hundreds of pulses per minute. The pacemaker electrode is connected to the *Pacemkr. Output* (pacemaker output). The pacemaker circuit is energized by switching the pacemaker *On-Off* switch to the *On* position. The *Defib. Pacemkr.* (defibrillator pacemaker) selector switch must be in the *Pacemkr.* (pacemaker) position. Each pacemaker pulse is indicated on the *Pacemkr.* lamp.

To turn the unit on preparatory to use, both the defibrillator and pacemaker pulse rate controls must be turned to 0 because the 0 stop is interlocked with the motor starting circuit. With these controls at 0, the start button is depressed for about 2 seconds and then released. The *On* lamp then lights. There is also a stop button to de-energize the unit.

Comment.—This instrument provides continuous variability of voltage, pulse width, and pulse frequency within known ranges of effectiveness for defibrillation. Optimal pulse characteristics may easily be obtained. Wide variability is available for experimentation. The pacemaker component utilizes pulse characteristics previously determined to be satisfactory and the pulse frequency is variable within a range suitable for all clinical and most experimental applications.

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TRACHEAL COMPRESSION CAUSED BY AN ANOMALOUS INNOMINATE ARTERY ARISING FROM A BRACHIOCEPHALIC TRUNK

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VASCULAR ANOMALIES of the superior mediastinum are surgically important because they may produce compression of the trachea and of the esophagus.[1, 2, 3] Anterior compression of the trachea without involvement of the esophagus may occur. It may be produced by mediastinal tumor, anomalous innominate artery, anomalous left carotid artery, and incomplete development of the tracheal cartilages. Gross[4] states that the left carotid artery may have a common origin with an anomalous innominate artery; this origin is from the distal portion of the arch of the aorta. Anterior compression of the trachea usually results in dyspnea, cyanosis, inspiratory and expiratory crowing, and recurrent pneumonitis because of inability to raise secretions.

The subject of this report is a case of anterior tracheal compression caused by an innominate artery originating from a brachiocephalic trunk. A review of the literature fails to reveal such a congenital anomaly causing tracheal compression.

CASE REPORT

An 8-month-old Negro female was admitted to the pediatric ward on Aug. 26, 1957, with a 2-month history of respiratory difficulty. The prenatal history was noncontributory. The child was a product of a normal delivery and weighed 6½ pounds at birth. She was in relatively good health for 2 months when she experienced an attack of chicken pox from which she recovered without difficulty. At the age of 2 months respiratory difficulty occurred, characterized by a definite expiratory wheeze and tugging respiration which were exacerbated on exertion and crying. The child did not cough and was able to eat well. Swallowing liquids at times was followed by severe cyanosis. On three occasions in the following 6 months the child experienced acute respiratory distress and cyanosis, which required hospitalization for periods of 2 to 3 weeks. X-ray studies at that time in another hospital were reported as being within normal limits. Following the third period of respiratory difficulty the child was seen in the outpatient department of the Jefferson Hospital and x-ray studies of the chest with barium swallow were performed. Lateral chest roentgenograms showed anterior tracheal compression just below the thoracic inlet.

Physical examination on admission to the hospital revealed a well-developed 8-month-old Negro female who was lying in the prone position with the head extended markedly on the trunk. The child was in obvious respiratory distress and a gross expiratory wheeze was

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present. Her temperature was 37.5° C., pulse 130, and repirations 40 per minute. Positive physical findings were confined to the thorax. Many coarse rhonchi were heard in the upper portion of the thorax and thought to be transmitted to the upper tracheobronchial tree. An expiratory stridor was present. Laboratory findings were as follows: hematocrit 32, hemoglobin 7.8 Gm. per 100 ml., white blood cell count 6,000, segmented neutrophils 36, lymphocytes 62, and monocytes 2. Urinalysis was within normal limits. Culture of the nose and throat revealed normal flora and bronchial cultures revealed an occasional staphylococcus. The serology was reported as negative. The injection of purified protein derivative, strengths No. 1 and No. 2, produced a negative response. Electrocardiogram revealed sinus tachycardia with no evidence of heart disease. X-ray studies of the chest showed a prominent thymic shadow. In the lateral views there was narrowing of the lumen of the trachea along its anterior margin just below the thoracic inlet (Fig. 1).

Fig. 1.

Bronchoscopy performed on Sept. 9, 1957, revealed constriction of the trachea just below the cricoid cartilage and this was thought to be due to extrinsic compression. The attending staff considered the pathologic condition to be due to an anterior mediastinal mass compressing the trachea.

On consultation, the surgeons considered the constriction of the trachea at the level of the thoracic inlet to be due to an anomalous vessel, either an anomalous innominate, or left carotid, artery. No roentgenologic evidence of mediastinal mass could be found. Surgical exploration and correction of the anomaly was recommended.

Exploratory thoracotomy through a left anterolateral incision was performed on Sept. 16, 1957, under endotracheal anesthesia. A small endotracheal tube was passed without difficulty. After dissection of the mediastinum it became apparent that we were dealing with a brachiocephalic vessel which originated from the distal aspect of the aortic arch. This common trunk gave off the innominate artery and the left common carotid artery 2.2

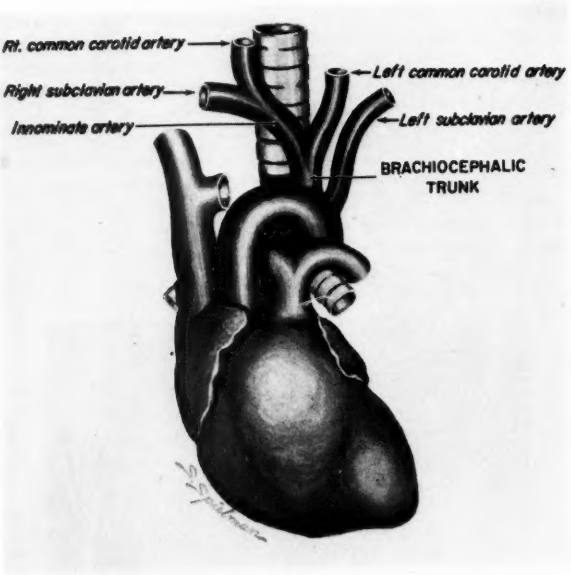
Rt. common carotid artery
Right subclavian artery
Innominate artery
Left common carotid artery
Left subclavian artery
BRACHIOCEPHALIC TRUNK

Fig. 2.

Fig. 3.

cm. from its origin on the aortic arch. The left subclavian originated approximately 0.5 cm. to the left of the brachiocephalic vessel (Fig. 2). The innominate artery followed a 45-degree course through the anterior mediastinum and passed anterior to the trachea just below the thoracic inlet. The entire brachiocephalic trunk, the innominate and carotid arteries were completely freed by sharp dissection. Three sutures were placed throughout the adventitia of the brachiocephalic trunk at its origin from the aortia. Two silk sutures were placed through the adventitia of the innominate artery as it crossed over the trachea. All these sutures were brought out through the sternum. This procedure pulled the anomalous innominate artery off the trachea and held it in satisfactory position.

The thorax was closed without drainage. The procedure was tolerated well and the patient was returned to the recovery room in good condition. After recovery from anesthesia, the child's respirations were obviously markedly improved. The respiratory stridor was no longer evident. The postoperative course was complicated by a rectal temperature elevation to 38.8° C. on the second postoperative day. Roentgenograms taken at this time showed atelectasis of the right upper lobe. A high humidity oxygen tent was utilized. The temperature returned to normal levels on the third day and the rest of the postoperative period was completely uncomplicated.

The patient was discharged on the twelfth postoperative day and was last seen 6 months after operation at which time the mother stated that the previous respiratory difficulty had been completely relieved. The child was entirely normal and showed satisfactory growth and development. Follow-up x-rays revealed complete relief of the tracheal obstruction (Fig. 3).

DISCUSSION

Anterior compression of the trachea, causing respiratory obstruction without involvement of the esophagus, may be caused by one of two vascular anomalies. An anomalous innominate artery may originate farther to the left on the aortic arch and pass anterior to the trachea in its course to the right thorax. Depending on its length and laxity, it may or may not cause enough narrowing of the trachea to produce respiratory obstruction.

An anomalous left common carotid artery originates from the right side of the aortic arch and also passes anterior to the trachea. It can cause obstruction in the same manner as an anomalous innominate artery.

The symptoms caused by these anomalies are referable to the respiratory tract. Symptoms of esophageal obstruction such as occur with double aortic arch and right aortic arch with a ligamentum arteriosum are absent, as the esophagus is not impinged upon. Recurrent, pulmonary infection, dyspnea, cyanosis, crowing respiration, and suprasternal tugging may be seen with this type of tracheal obstruction. The patient may assume a characteristic position; a prone position with hyperextension of the neck seems to improve expiratory exchange. Flexion of the head on the trunk exacerbates respiratory distress.

Lateral roentgenograms of the chest will reveal anterior narrowing of the tracheal air column at its lower third. Tracheograms performed with opaque media will more clearly delineate the site of compression. Tracheograms were not performed on our patient, since the site of compression was readily detected on regular lateral chest films. Angiocardiography or retrograde aortography may be of great value in the rare case, but is thought to be usually an unnecessary diagnostic procedure.

Endoscopy may be helpful in viewing the site and determining the cause of the tracheal compression. At times, tracheal compression due to vascular

anomalies may cause transmitted pulsations which are readily visible through the bronchoscope. Compression due to incomplete development of the tracheal cartilages can be determined by endoscopic examination as the caliber of the tracheal lumen will decrease on expiration.

Anterior compression of the trachea may also be caused by a mediastinal mass. Lateral x-ray views of the chest should then reveal an anterior opacity impinging on the trachea.

EMBRYOLOGY

The formation of a brachiocephalic trunk in the human being has not been explained. The brachiocephalic trunk presents as a part of the adult circulation in the sheep and dog.

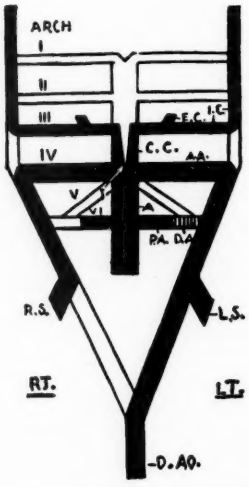

Fig. 4.—Coarctation of the aorta showing diagrammatic sketch of the aortic arches. Blocked areas represent the definitive arteries, unblocked areas those that disappear.

I, II, III, IV, V, and *VI* are the arches. *E. C.*—External carotid. *C. C.*—Common carotid. *I.*—Innominate. *A. A.*—Aortic arch. *A.*—Aortic sac. *P. A.*—Pulmonary artery. *D. A.*—Ductus arteriosus. *R. S.*—Right subclavian. *L. S.*—Left subclavian. *D. Ao.*—Descending aorta. (*From* Herbut: Surgical Pathology, ed. 2, Philadelphia, 1954, Lea & Febiger.)

In the human being, the aortic sac, its left horn, and the left fourth arch artery form the arch of the definitive aorta (Fig. 4). The right horn of the aortic sac elongates to become the innominate artery. The proximal part of the left third arch artery becomes the left common carotid artery.[5]

We postulate that the failure of formation of cleft in the aortic sac labeled *A* in the diagram (Fig. 4) would cause the development of a common trunk between the innominate artery labeled I and the left carotid artery labeled *C. C.* The pull of the left common carotid at its origin from the trunk might also explain the position of the brachiocephalic trunk on the left of the midline.

SUMMARY

1. A patient with anterior tracheal compression by an anomalous innominate artery originating from a brachiocephalic trunk is reported.

2. The surgical correction of the anomaly is detailed.

3. A possible explanation of the embryologic development of this anomaly is suggested.

The authors wish to thank Peter A. Herbut, M.D., Professor of Pathology of the Jefferson Medical College, for permission to use Fig. 4 in this article and for his advice on the developmental anatomy of this anomaly.

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TRAUMATIC RUPTURE OF THE ESOPHAGUS

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SINCE Barrett's[1] description of the successful repair of a postemetic perforation of the esophagus in 1946, at least 200 cases have been reported, describing the primary surgical attack, and successful repair of the lesion. Mechanism of rupture has evoked considerable discussion and the advancement of many theories. Many anatomic and physiologic studies have been performed on the esophagus in an attempt to prove the method of perforation. It has been noted that perforation almost invariably occurs in the left lateral posterior aspect of the esophagus, several centimeters above the hiatus. Mackler,[2] in a study of 65 autopsy specimens, ligated the cardia of the stomach and the proximal portion of the esophagus. He then distended the specimens with air and noted that the esophagus perforated in a longitudinal direction in the left lateral posterior position, in all but 3 cases. Mosher[3] has described the anatomic features of the lower esophagus which probably contribute to rupture in this area. He noted that the circular smooth muscle of the lower esophagus becomes longitudinal close to its junction with the stomach and fans out over the stomach to help form the muscular layers. The longitudinal direction of the fibers allows easy separation, so that the mucosa, which is unsupported with a submucosal layer, can herniate through and burst. Mackler, in his study, noted that muscle fibers split first, the mucosa then bulges through the separation and ruptures, thus explaining the longitudinal character of ruptures in this area. The basic theory of mechanism, which appears most reasonable, is that the forceful ejection of gastric contents into the anatomically weak lower esophagus, during the process of vomiting, distends the lower esophagus to the point of rupture. A case report of an individual, who was struck in the epigastrium immediately after a meal with a resulting rupture of the lower esophagus, is presented because it appears to lend credence to the theory that rupture is caused by a transmission of force from the stomach into the lower esophagus, via the suddenly ejected gastric contents.

CASE REPORT

A 46-year-old rancher (H.H.) was admitted to the hospital with a history of having been crushed between two trucks, immediately following a hearty breakfast. The patient

From the Donahoe Clinic, Sioux Falls, S. D.
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was struck in the epigastrium and back. He states that immediately on contact his breakfast "shot out of his mouth" and he developed severe lower chest and back pain. On admission to the hospital, it was noted that the patient was dyspneic and in acute distress, complaining of an excruciating epigastric and back pain.

Physical examination* revealed a normal temperature, a pulse of 100, and a blood pressure of 110/70 mm. Hg. A hematoma was noted over the lumbar spine. There was rigidity in the epigastrium, and the remainder of the abdomen was tender. During the

Fig. 1.—Spot film taken during fluoroscopy. Arrow marks site of esophageal perforation.

next few hours, the patient continued to complain of shortness of breath and pain. The tenderness and rigidity of the abdomen diminished. Because of continued deterioration in the general condition of the patient, a swallow of barium was given, and a roentgenogram of the chest was made. It was noted that barium had escaped into the area lateral to the esophagus, above the left diaphragm. The patient was transferred to Sioux Falls for surgical therapy, with the provisional diagnosis of rupture of the diaphragm, or rupture

*By Dr. R. W. Roesel of Burke, South Dakota.

of the lower esophagus. Upon admission to the hospital in Sioux Falls, a swallow of Lipiodol was given, and the site of perforation was noted (Fig. 1). Sixteen hours after injury, the chest was opened through the bed of the ninth rib. Serosanguineous fluid was noted in the thoracic cavity. The mediastinum was markedly distended with most of the distention present in the inferior pulmonary ligament. The mediastinal pleura was opened by cutting the inferior ligament up to the inferior pulmonary vein. A large amount of gastric content was removed from the mediastinum. Perforation was located in the posterior lateral aspect of the esophagus, immediately above the hiatus, and measured 1.5 cm. in length. The mediastinum was irrigated with quarts of normal saline, and an extensive débridement was carried out. The laceration was closed with interrupted 3-0 silk sutures. The chest tube was sutured to the diaphragm, in close proximity to the perforation, and brought out the posterior axillary line in the ninth inner space.

The patient's postoperative course was relatively uneventful. Three hundred cubic centimeters of serosanguineous fluid was aspirated from the right thorax. On the fifth day the temperature was normal. On the seventh day, the Levin tube was removed and the patient was started on a clear liquid diet. On the tenth day, the patient was advanced to a full liquid diet which, by the fourteenth day, was changed to a soft diet. The chest tube was removed on the fourteenth day. Since this time, the patient has returned to his work and has had no further complaints.

DISCUSSION

A review of the literature reveals 6 cases[2, 4-8] of rupture of the lower esophagus, following nonpenetrating trauma to the epigastrium and lower chest. Five cases died without a diagnosis, the sixth[8] survived with closed drainage. It is believed that the forceful ejection of the patient's breakfast from his mouth immediately upon being struck by the truck is significant since it demonstrates the rapid emptying of gastric contents into the esophagus. Most cases of postemetic rupture of the esophagus occur when the patient vomits following ingestion of a large meal, or a heavy drinking bout. Because the rupture in the above case was identical with the type known as postemetic perforation, one may assume that similar etiological factors are present in the two groups of cases. Both have in common the forceful ejection of gastric contents into the anatomically weak lower esophagus. The lack of survivors following indirect trauma denotes a general unawareness of this type of chest injury. Early definitive treatment gives good results, as evidenced by the large number of successfully repaired postemetic esophageal ruptures. An awareness of the possibility of esophageal perforation from nonpenetrating injury will certainly lead to the early discovery of the lesion, since diagnosis is easily accomplished by simple radiologic procedures.

SUMMARY

A case of rupture of esophagus due to a blow upon the stomach is reported (1) because of the light it throws on postemetic rupture of the esophagus, and (2) because of its successful treatment by primary repair.

Both types of rupture have in common the sudden forceful ejection of gastric contents into the anatomically weak lower esophagus.

Early diagnosis by roentgenologic studies is mandatory.

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EXPERIMENTAL USE OF NYLON MESH FOR ESOPHAGEAL SUPPORT

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THE SUCCESSFUL clinical use of fine mesh nylon for support of a markedly weakened segment of esophageal wall and for the satisfactory function of this repair, for 3 years following operation, has prompted this investigation relative to the suitability of fine mesh nylon for esophageal support.[10]

The usefulness of fine mesh nylon in providing needed support for anatomically weakened structures in various parts of the body, both experimentally and clinically, is well recorded in medical literature. The clinical utilization of nylon mesh in the repair of abdominal wall hernia defects was first suggested and carried out by Aequaviva and Bourret[1, 2] in 1944. Since that time, a number of clinicians have reported the successful use of nylon mesh in hernia repair.[4, 5, 7, 13, 15, 16, 17] Testa,[20] in 1951, was the first to study the suitability of nylon mesh in the repair of experimentally created defects in animals. He employed a wide-mesh monofilament nylon with success in the repair of musculofascial abdominal wall defects in rabbits. The senior author and Siderys,[11] in 1955, used fine-mesh nylon experimentally to repair large abdominal wall defects in dogs. Satisfactory repairs were obtained and the nylon mesh was found to be tolerated well, both on gross and microscopic examination.

Nylon mesh had been employed with success in obtaining anatomic support in other areas. Vargas and Deterling[21] have described the use of nylon net in the external support of blood vessel grafts and aneurysms. The use of nylon mesh in the repair of diaphragmatic hernias has been reported by Adler and Firme.[3] Porell and Parsons[14] have recently recorded the successful use of nylon mesh in 2 cases in the repair of troublesome perineal hernias which developed as late sequelae of pelvic exenteration.

Although impervious synthetic tubes have been employed both experimentally and clinically for esophageal replacement, the authors have been unable to discover any published experience with the use of fine-mesh nylon for esophageal support. For this reason it has seemed desirable to record our experience with the use of fine-mesh nylon in the repair and support of partially resected segments of the wall of the esophagus.

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MATERIAL AND METHODS

Healthy mongrel dogs were used. A mixture of oxygen and ether, delivered through an endothracheal tube by means of a mechanical respirator, was employed for anesthesia following induction with intravenous Pentothal. A left sixth intercostal space thoracotomy incision was employed.

When the left chest was opened, the lower third of the esophagus was dissected free from surrounding structures. Straight Potts coaretation clamps were used to cross-clamp the upper and lower portions of the freed-up segment of esophagus. The Potts clamps prevented bleeding from the esophageal musculature during its extramucosal resection and greatly facilitated the dissection by providing a bloodless field. They also made easier the exposure and manipulation of this segment of esophagus. Esophageal injury did not appear to have resulted from the use of these clamps in any of the animals studied.

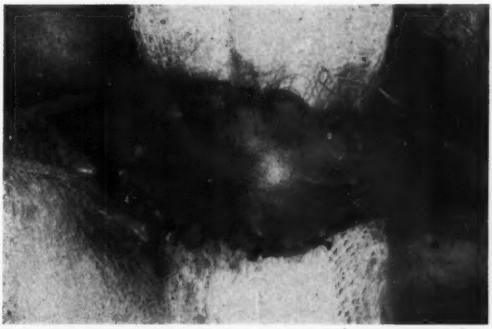

Fig. 1.—Photograph of the esophagus, at the time of operation, showing the appearance of the fine-mesh nylon patch immediately after its placement.

With an area of esophagus secured between Potts coaretation clamps, an oval segment of esophageal musculature was resected. The denuded areas of esophagus averaged 2.5 by 3.5 cm. in size. It soon became apparent that relatively little weakness in the wall of the lower esophagus of the dog was produced by an extramucosal resection of muscle in this area. The mucosa and submucosa of the lower portion of the canine esophagus are rather thick and possess a surprising degree of strength. The defect which had been created in the wall of the esophagus was repaired by suturing in place, with some slight overlap, a patch of sterile nylon mesh (241-F).* The nylon mesh contained 270 by 204 threads per square inch. Interrupted sutures of 4-0 or 5-0 silk were employed (Fig. 1). The Potts clamps were released when the nylon mesh patch was secured.

The chest wall was closed in layers and a catheter which was left in the chest during the closure was aspirated and withdrawn. Esophageal defects

*The nylon was obtained from Stehli Company, Inc., 1372 Broadway, New York, N. Y.

were created and repaired with fine-mesh nylon patches in this manner in 11 dogs. In 4 additional dogs, defects were created, but were left unrepaired, to serve as controls.

RESULTS

All 4 of the control dogs were sacrificed between 3 and 4 months following operation. There was no evidence in any of the animals of bulging or distortion of the esophagus or of ischemic or necrotic change in the area of muscle resection. Weakness of the esophageal wall in the area of the muscle defect could not be demonstrated by distention of the esophagus with water under pressure. From these observations, it was apparent that significant

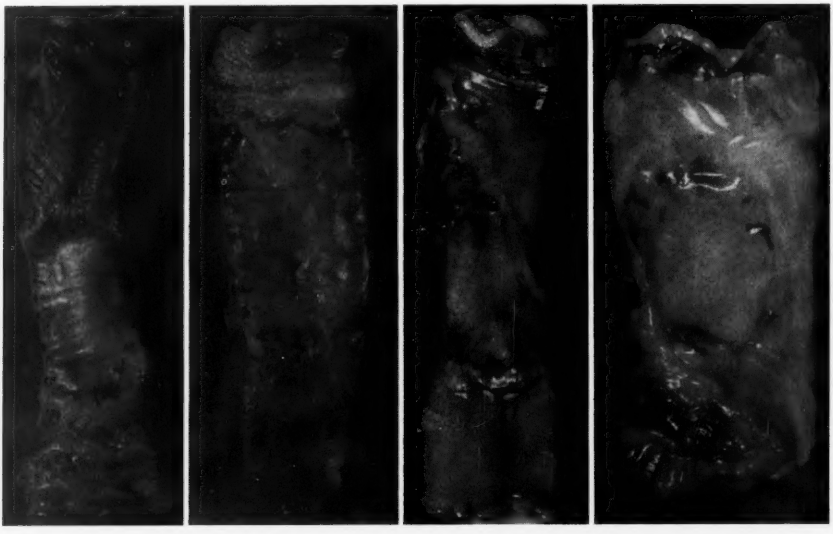

Fig. 2.—Photograph of segments of esophagus, taken at necropsy, demonstrating the satisfactory tissue tolerance and retention of shape and position of the nylon patch 5 and 6 months after operation.

esophageal weakness could not be produced by extramucosal resection of the esophageal musculature in the dog. It was also of interest that rather extensive resection of the musculature could be carried out without the development of ischemia or other disturbances in the underlying structures.

The fine-mesh nylon appeared to be tolerated quite well in all 11 animals in which it was used to replace resected esophageal musculature (Fig. 2). Post-mortem studies of the segment of esophagus containing the nylon mesh patch were carried out 1 month following operation in 2 animals and 4 months following operation in 1 animal. Eight of the animals were sacrificed between 5 and 6 months after operation. No areas of esophageal weakness could be demonstrated at autopsy and no bulging could be demonstrated

in the "supported" area by distending the excised segment of esophagus with water under pressure. A remarkable absence of gross reaction in the surrounding tissue to the presence of the nylon patch was observed. In all

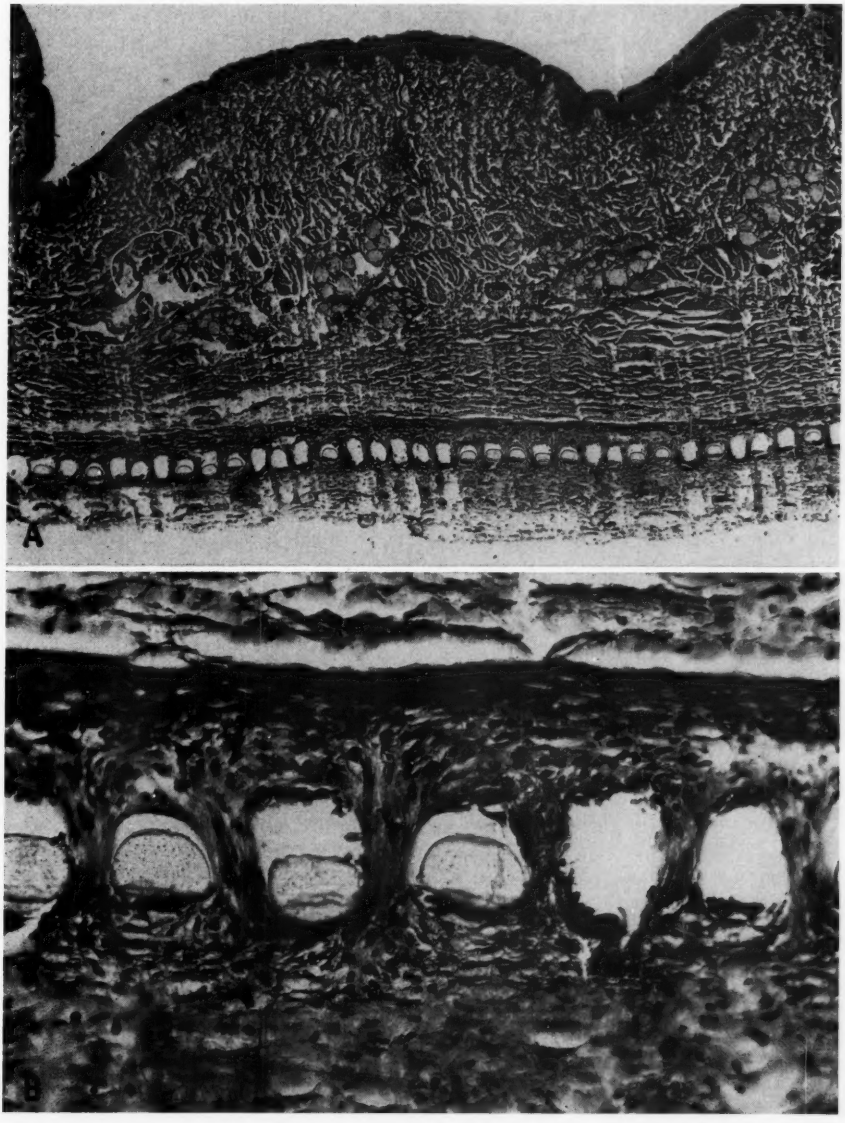

Fig. 3.—Photomicrographs, under high and low power, illustrating the excellent manner in which the nylon mesh was tolerated and incorporated into the structure of the wall of the esophagus 5 months following operation.

of the animals, the nylon patch and all of its securing interrupted silk sutures had remained intact and in place. There was no evidence of fluid accumulation, encystment, or infection in any of the animals. A minimal degree of transverse wrinkling of the fine-mesh nylon was encountered in 1 of the animals. There was no wrinkling of the nylon patch in the 10 remaining dogs. Neither scarring nor constriction of the esophagus were noted at the site of graft placement in any of the animals. The esophageal mucosa in the areas supported by the fine-mesh nylon graft was normal in all of the animals.

Microscopic study of the areas of nylon replacement of esophageal musculature revealed excellent tolerance of the nylon patch (Fig. 3). Virtually no reaction in the tissue surrounding the foreign body was encountered. The nylon patch in all of the animals appeared to be firmly incorporated into the surrounding tissue through the invasion of its interstices with fibroblasts. There was no evidence of gross or microscopic damage which might have resulted from the use of the Potts coaretation clamps for hemostasis and manipulation of the esophagus during operation.

DISCUSSION

From a review of the literature, it is apparent that little harm can come from leaving a small area of submucosa bare of muscular covering. Small areas of mucosa are frequently and successfully left bare intentionally in other portions of the alimentary tract, especially in performing cardiac and pylorie myotomies at the extremities of the stomach. In the dog with its thicker and more substantial esophageal mucosa, even larger areas of submucosa, as this study would seem to indicate, may be left barren of muscular covering and support with safety.

The principal published clinical experience with extramucosal resection of segments of the wall of the thoracic esophagus concerns the surgical management of benign tumors of the esophagus, mainly leiomyomas.[6, 8, 18, 19] In most cases, the defect following removal of a leiomyoma has been repaired successfully by approximating the adjacent esophageal muscle over the defect. Occasionally the remaining mucosa is quite thin and further steps may be required to prevent its subsequent destruction. In 2 of the 11 cases reported by Harrington,[8] extremely thin areas of remaining mucosa were incised and the remaining mucosa was closed with the reconstruction of the outer wall of the esophagus. However, when larger extramucosal defects occur it is apparent that such means cannot be resorted to without accepting the risk of significantly narrowing the lumen of the esophagus. It is also desirable to avoid entering the esophageal lumen whenever possible. Mucosal difficulty has been reported following extramucosal resection of benign esophageal tumors. Meade,[9] in discussing the paper of Sweet and his associates,[19] reported that one of two extramucosal resections was complicated by disintegration of the mucosa and the death of the patient. It is possible that other difficulties may have occurred, but have escaped publication.

Benign esophageal tumors, on occasion, may completely encircle the esophagus and may involve a rather considerable length of this structure. Nissen,[12] in 1949, reported the extramucosal removal of a large, circumferential leiomyoma from 10 cm. of the esophagus. The rather lengthy segment of barren mucosa was wrapped in a pedicle flap of lung tissue and the patient survived. Other measures, such as the use of pleural flaps, have been reported for the support of weakened areas of esophagus.

Although significant weaknesses in the esophagus wall could not be produced by the techniques of extramucosal resection employed in this study, as illustrated by the experience with the control animals, the ability of nylon mesh to support areas of anatomic weakness in the human body is well established. It may be assumed that this ability would be as effective in supporting esophageal weaknesses as those occurring elsewhere. One of the more important facets of this study was the demonstration of the tolerance of the esophagus for the nylon mesh and the capacity of the nylon mesh to remain intact and in place, despite the movement and changes in size to which the esophagus is subjected during deglutition. The nylon mesh was tolerated well in the wall of the esophagus, both grossly and microscopically. Wrinkling seldom was encountered. There was no fibrosis or contraction of the nylon graft and neither stricture or narrowing of the esophageal lumen was encountered. The appearance of the underlying mucosa remained normal in all cases. Neither fluid accumulation, encystment, nor infection occurred.

The cross-clamping of the esophagus above and below the area of extramucosal resection with straight Potts coaretation clamps proved to be a valuable aid in the handling of the esophagus and in permitting the dissection to be carried out in a bloodless field. This maneuver appeared to be tolerated well and was not associated with any early or late untoward effects.

SUMMARY

The suitability of nylon mesh in providing support for anatomically weakened structures is well established. The use of fine-mesh nylon in the replacement of resected extramucosal segments of esophagus in 11 dogs is reported. The nylon mesh was tolerated quite well and remained securely attached to the margins of the artificially created defect throughout the periods of observation in all of the animals studied. There was complete freedom from scarring, encystment, and infection in all. The use of fine-mesh nylon for added esophageal support when needed is recommended.

The use of noncrushing, multitoothed Potts coarctation clamps for handling the esophagus, and permitting work upon it in a bloodless field, is described.

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BRONCHOPULMONARY MONILIASIS

Treatment by Segmentectomy

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BRONCHOPULMONARY moniliasis, an infection of the lungs by *Candida albicans*, has been recognized for over half a century. Hurwitz and Yesner[1] reported the first successful surgical extirpation of localized bronchopulmonary moniliasis in 1947. In spite of the increasing frequency with which circumscribed pulmonary lesions of obscure origin are excised as a diagnostic and therapeutic maneuver, no other reports of successful surgical treatment of this unusual pulmonary disease have been found in the literature. It is the purpose of this report to record a second successful surgical management of bronchopulmonary moniliasis.

CASE REPORT

A married white man, a native of New York, 35 years of age, was admitted to Scripps Memorial Hospital, La Jolla, Calif., on Nov. 14, 1956, complaining of recurrent hemoptysis for 2½ years. There was no history of exposure to tuberculosis. The patient was stationed in the southwestern region of the United States during the Second World War until his separation from the military service on a medical survey because of essential hypertension. Five years previously, following a trip to Nevada, the patient noticed an upper respiratory infection with fever and nonproductive cough, following which a photofluorogram at a public health department facility showed infiltration in the left mid-lung field in which a cavity subsequently appeared. Intradermal injection with purified protein derivative, U.S.P., 0.1 mg., and coccidioidin, 0.1 c.c. 1:100 dilution, produced a positive reaction. Cultural examinations of serial sputa were negative for tubercle bacilli and fungi at this time. The patient was observed for 9 months with a presumptive diagnosis of cavitary coccidioidomycosis without evidence of change in status.

The patient had smoked forty cigarettes per day for over 10 years and noted a cough productive of approximately 3 c.c. of grayish white mucoid sputum each day which contained small black particles. He reported dyspnea on climbing one flight of stairs.

In 1953, because of recurrent hemoptysis, the patient had been hospitalized, at which time bronchoscopy and bronchography were reported to show no abnormality. Over the ensuing 2½ years, the patient noticed gross hemoptysis, often lasting one day, and accompanied by some left anterior chest pain, occurring approximately once per month.

The family history and occupational history were not remarkable, and the review of systems was otherwise noncontributory.

Physical examination revealed a well-nourished man who did not appear ill; weight 163 pounds, height 68.5 inches, temperature 98.6° F., blood pressure 150/80 mm. Hg, heart

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rate 80, and respirations 20. Other than for a small crusting lesion, resembling epidermophytosis, posterior to the medial malleolus of the right ankle, the physical examination was entirely normal.

Examination of the urine revealed no abnormalities; hemoglobin was 14 grams per cent, the erythrocytes numbered 5.2 million and leukocytes 6,400 per cubic millimeter with a normal peripheral blood smear. Cultures for fungi taken of skin scrapings from the scaling lesion of the right foot showed no mycotic forms in 1 month. There were no tubercle bacilli in the sputum in cultural examination of three specimens, and three specimens of sputum of the same unusual character with small black particles in it grew pure cultures of *Candida albicans* on modified eosin-methylene blue medium after the method of Weld[10] (San Diego County Department of Public Health Laboratory). A complement fixation test for *Coccidioides immitis* was 3-plus in the 1:2 dilution, which was thought to be nondiagnostic. A pulmonary ventilation study was normal before and after a bronchodilator.

Fig. 1.—Preoperative roentgenogram of the chest. A cavity can be seen in the left mid-lung field.

In a roentgenogram of the lungs (Fig. 1), there appeared a cavity in the left mid-lung field which in the lateral view appeared to lie in the superior segment of the left lower lobe. Planograms failed to reveal calcification in the wall of the cavity and showed no other associated lesions. Preoperative bronchoscopic examination revealed no abnormality.

Surgical Procedure.—On Nov. 15, 1956, a left thoracotomy was performed through the fifth intercostal space. There was no free fluid, nor any evidence of pleural disease. At the apex of the left lung there were multiple small bullae. In the superior segment of the left lower lobe was a thin-walled cavitary lesion which could be felt through normal lung tissue, and scattered about it were a number of fibrocalcific lesions. A superior segmentectomy was carried out with ligation of the segmental vessels and interrupted suture closure of the segmental bronchus. The raw lung surfaces were closed with interrupted sutures of 4-0 silk. Anterior and posterior catheter drains were placed in the second anterior

and the eighth posterior interspace and connected to water-seal drainage. The patient received 2,500 c.c. of whole blood during the procedure, and the thoracic incision was closed in layers with silk.

Postoperatively, the patient's convalescence was uneventful. The gross pathologic specimen is shown in Fig. 2, and there can be seen a thick-walled cavity measuring 3 cm. in maximal diameter. The wall averaged 2 to 3 mm. in thickness and was firm and grayish white. Microscopic examination (Thomas A. Le Strange, M.D.) showed the above-described cystic lesion to be partially lined with bronchial mucosa showing foci of squamous metaplasia. Communication with a bronchiole is demonstrated in Fig. 3. The majority of the cystic area was lined by fibrous tissue, into which there was a marked chronic inflammatory cellular infiltration. The fibrous tissue was covered with a hemorrhagic exudate. Within the cavity of the lesion, definite spore forms with mycelia were demonstrated with periodic acid-Schiff stain technique. These fungi were consistent with *Candida albicans*. The adjacent pulmonary tissue surrounding the cystic lesion showed marked scarring, atelectasis, and chronic inflammatory changes. Multinucleated cells, although not numerous, were

Fig. 2.—The resected superior segment of the left lower lobe. The arrow indicates the cavity.

demonstrated. A high-power view of mycelia demonstrated with periodic acid-Schiff stain technique is shown in Fig. 4.

Four months postoperatively, although the patient felt well and had returned to work and had noted no further bleeding episodes, he noticed a return of small amounts of mucoid sputum containing small black particles. Culture of the sputum was again positive for *Candida albicans*. As at that time there was no other effective fungicidal agent available, the patient was treated with inhalation therapy using a bronchodilator and an aerosol of brilliant green dye in a 1:200,000 dilution by positive pressure therapy for 1 month.

The patient has now been well 1 year, raises no sputum, has had no further bleeding episodes, and the small amount of mucoid secretion which the patient raises from his bronchial tree shows no cultural evidence of fungi.

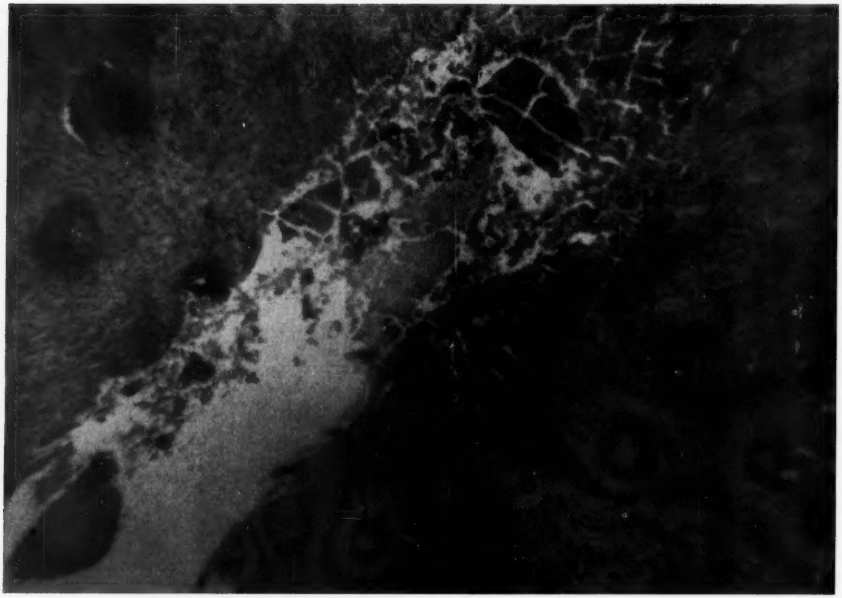

Fig. 3.—Low-power photomicrograph demonstrating communication of a cavity with a bronchiole.

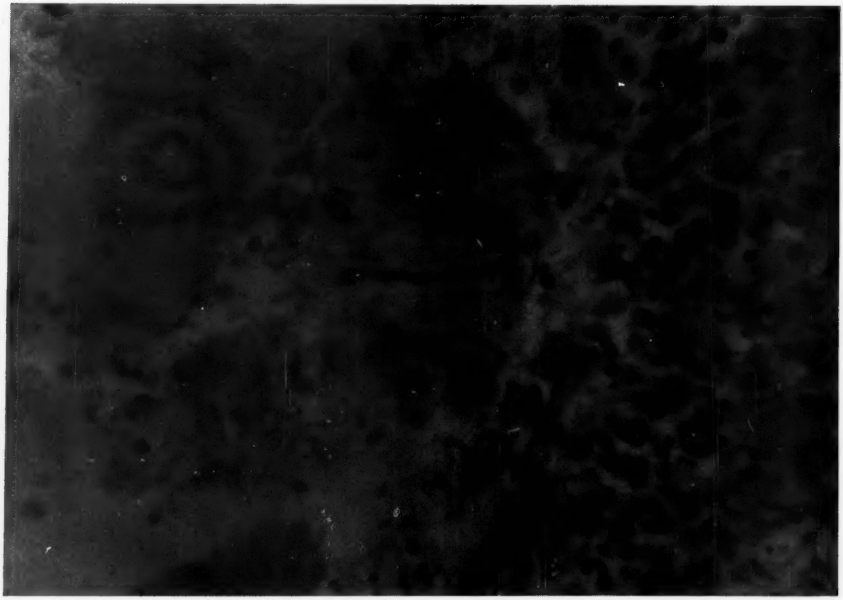

Fig. 4.—A high-power photomicrograph of the same area showing mycelia consistent with *Candida albicans*. (Periodic acid-Schiff stain.)

DISCUSSION

Pulmonary moniliasis is not as rare a disease as is generally assumed, being next in frequency to actinomycosis among the mycotic diseases of the lung.[11] Although *Candida albicans* is a saprophyte present in the body in health, it has been shown to have the ability to produce an invasive and occasionally fatal pulmonary infection.[2, 6, 9] The medical management of this disease has been difficult and variable. Kass, Sasano, and Klein[5] reported successful treatment of one patient with brilliant green aerosol inhalation. Successful treatment with brilliant green aerosol has likewise been reported by Smith and Arman,[8] in 1955. More recently Scott[7] has reported a possible case of pulmonary moniliasis involving both lungs with dramatic response to potassium iodide therapy and has discussed the advent of a new fungicidal agent, Amphotericin B.

SUMMARY

A case of symptomatic cavitary bronchopulmonary moniliasis successfully treated by segmentectomy and postoperative brilliant green aerosol therapy has been reported. It is believed that this is the second reported successful surgical treatment of this disease.

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SOLITARY RHABDOMYOSARCOMA OF THE PLEURA

Report of a Case With a Note on the Nomenclature of Pleural Tumors

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ONE of the features of striated muscle tumors is their frequent occurrence in sites in which striated muscle is either scanty or absent. This fact has led to much speculation on the tissue of origin of rhabdomyomatous tumors which arise in such places. A neoplasm illustrating this aspect of abnormal muscle cell growth is seen in the following case, which is also remarkable for the similarity of the clinical and gross pathologic findings to those found in many cases of solitary fibroblastic pleural tumor.

CASE REPORT

Clinical History.—A male retired plumber, aged 69 years at the time of his death, was first seen 3 years before this, when his main complaints were recurrent chest pain and increasing breathlessness on exertion. These symptoms had become increasingly severe since an attack of an "influenza-like" illness some 4 years before. He also maintained that he was now getting bouts of fever with shivers and he had also noticed some pain and swelling in the joints of the fingers. Physical examination revealed absent breath sounds and diminished movement over the left side of the chest and a moderate degree of hypertrophic pulmonary osteoarthropathy of the fingers and toes. A chest radiograph and subsequent fluoroscopic examination showed a large opacity with a smooth, rounded upper border in the left side of the thorax with displacement of the cardiac shadow to the right. No fluid was obtained on attempted thoracentesis and this, combined with the radiographic appearances, prompted the radiologist to suggest the diagnosis of a benign intrathoracic tumor. Bronchoscopy revealed no abnormality. Fluid aspirated from the bronchi was negative for malignant cells and numerous subsequent sputum examinations also proved negative for both malignant cells and tubercle bacilli. Clinically, he was considered to have an inoperable carcinoma of the lung and a course of palliative deep roentgen therapy was instituted. Then followed a period of 2 years, during which he had intermittent courses of this therapy to the chest lesion, with some subjective improvement after each treatment. However, as the radiographic signs appeared to be increasing while the patient's general condition remained much as before, the diagnosis of carcinoma of the lung appeared open to doubt. The radiologist felt that the appearances were still those of a tumor rather than an inflammatory lesion, but as no alternative treatment was suggested, it was decided to persist with the deep roentgen therapy.

Increasing exertional dyspnea now began to incapacitate the patient and he was readmitted to hospital, 2 years and 5 months after his initial visit. At this time, he was

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noted to have marked clubbing of the fingers and toes, he was dyspneic on the slightest exertion and the chest radiographs showed a total opacity of the left side of the thorax. With bed rest, the patient's condition improved sufficiently for him to be discharged from the hospital, but he was readmitted 2 months later, again complaining of severe exertional dyspnea. He now had dyspnea even at rest and he had noticed swelling of the feet and angles. Slight cyanosis was evident and he had a mild pyrexia. A pleural biopsy was considered but dismissed because of the patient's poor general condition. Death occurred promptly. The total duration of symptoms was approximately 7 years.

Autopsy Findings.—Autopsy was performed 13 hours after death. Gross hypertrophic pulmonary osteoarthopathy of the fingers and toes was noted on external examination and subsequent dissection of the involved joints confirmed this. Pitting edema of the feet and ankles was present. As was anticipated, all the findings of importance were confined to the thorax. Here, there was a gross mediastinal shift to the right due to a large tumor in the left pleural cavity. This tumor measured 30 by 15 by 12 cm. in its largest diameters and it lay quite free in the pleural space, the visceral and parietal pleurae being quite simply separated from the tumor mass. Several fibrous strands ran from the pleural surfaces into the tumor, these being most marked over the area of the fourth, fifth, and sixth ribs just anterior to the costovertebral junctions. Adhesions were also seen over the lower portion of the neoplasm anteriorly, where fibrous strands attached it to the visceral pleura.

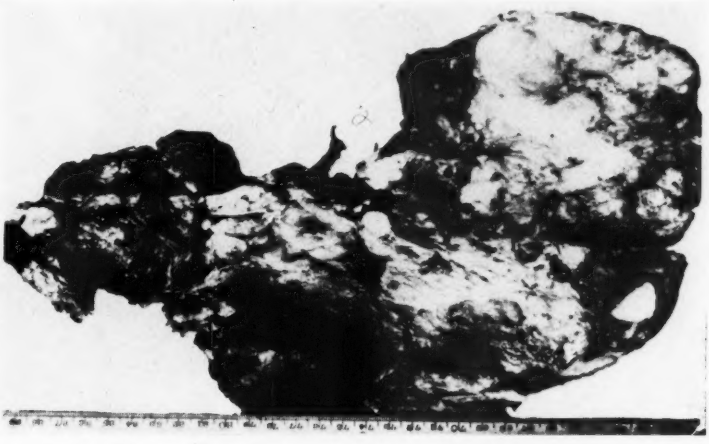

Fig. 1.—Cut surface of tumor. Scale in centimeters.

No definite point of origin of the tumor from surrounding structures could be demonstrated and no connection with the intercostal nerves or the sympathetic chain was noted. The tumor weighed 2,694 grams and appeared to be quite encapsulated, except in its superolateral portion where extensive necrotic change had taken place. The capsule was smooth and glistening and showed quite distinctly the smooth lobules of underlying tumor tissue. The cut surface of the tumor (Fig. 1) revealed a mass composed of firm nodules of tissue which varied in color from pale pink to grayish white, some of the nodules being up to 3 cm. in diameter. Whorls and trabeculae of what appeared to be fibrous tissue could be seen encircling and passing through the tumor masses. Numerous soft yellow areas of necrosis with occasional areas of hemorrhage were also seen and in the upper portion of the tumor this had progressed to almost complete liquefaction necrosis. This was thought to be the effect of the deep roentgen therapy. At no point was any invasion of the capsule seen and no invasion of any of the underlying tissue could be demonstrated. The pleural cavity contained approximately 900 ml. of dark hemorrhagic fluid, due to the liquefaction necrosis of the upper portion of the tumor.

The underlying left lung was collapsed and the bronchi grossly infected, while the right lung also showed areas of collapse and bronchopneumonia. Dissection of the pulmonary bronchi was carried to their finest radicles but no tumors could be demonstrated. Some atherosclerosis was present in the vascular system, the liver showed evidence of passive venous congestion, and some enlargement of the prostate was seen. No other tumors were found at autopsy despite meticulous search.

Microscopic Findings.—Microscopic examination of a number of organs and tissues including the thoracic tumor was undertaken, the material being fixed in 10 per cent formol saline. Representative blocks were cut and stained by a variety of methods, those used being Mallory's phosphotungstic acid hematoxylin, Crossman's modification of Mallory's connective tissue stain, van Gieson, Masson's trichrome technique, Foot's reticulum stain, and hematoxylin and eosin. The pulmonary tissues showed areas of atelectasis with much pyogenic infection and necrosis but no areas of neoplasia. The prostate was subjected to meticulous search but benign lobular hyperplasia only could be demonstrated. The liver showed some venous congestion. The pleural tumor exhibited a quite extraordinary appearance and multiple blocks were studied, all the above-staining techniques being employed. The most striking feature was the presence of areas of large eosinophilic cells with granular cytoplasm, many of them multinucleate and showing a variety of forms (Fig. 2). Round, triangular, and elongate cells were seen, some, 40 to 50 microns in diameter, the majority

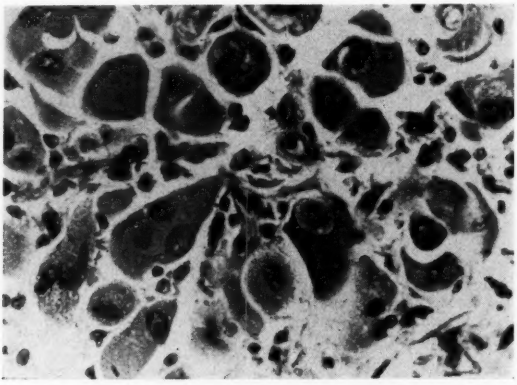

Fig. 2.—Collection of large granular eosinophilic myoblasts. (Hematoxylin and eosin; ×150; reduced ¼.)

having between 2 and 4 nuclei. These nuclei were generally eccentrically placed in the cell and exhibited prominent nucleoli with a fairly loose chromatin pattern and usually a perinuclear halo. The tissues appeared edematous and this seemed to have produced some separation of the cells. About and among these areas of large eosinophilic cells, many straplike and spindle-shaped cells could be seen, some fairly short and plump while others were slender, threadlike cells. Many of these cells appeared to be composed of longitudinal fibrils and on close examination in well-differentiated preparations many of the cells were seen to show definite cross striations (Figs. 3 and 4). As the figures show, these cross striations were demonstrated with equal facility by the phosphotungstic acid hematoxylin technique or the routine hematoxylin and eosin preparations. Connective tissue stains revealed a well-developed fibrous stroma. Large areas of the tumor appeared to consist solely of hemorrhagic, necrotic material and, in these areas, cellular detail had been lost, often the sole surviving cells being a few fibroblasts.

These large granular eosinophilic cells had the appearance of muscle tumor cells and the finding of cells composed of longitudinal fibrils, which exhibited definite cross striation,

confirmed the histologic diagnosis of a tumor of striated muscle. The final diagnosis was rhabdomyosarcoma of the left pleural cavity with atelectasis of the left lung and bronchopneumonia of both lungs.

DISCUSSION

Rhabdomyomatous tumors of the thoracic wall have been described by Stout[12] and Capell[2] and they have also been described within the substance of the lung.[10] Kirshbaum[8] and Perry and Smith[11] have reported the occurrence of malignant striped muscle tumors in the diaphragm, which spread to

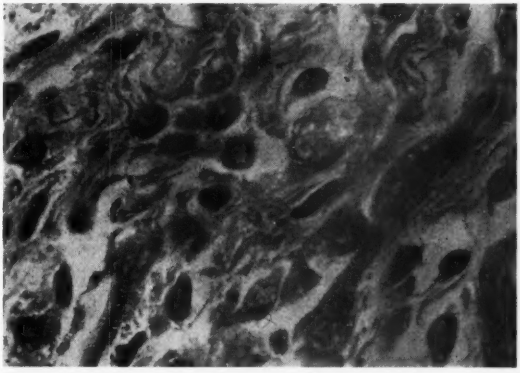

Fig. 3.—Well-defined cross striations in straplike cells. (Hematoxylin and eosin; ×400; reduced ¼.)

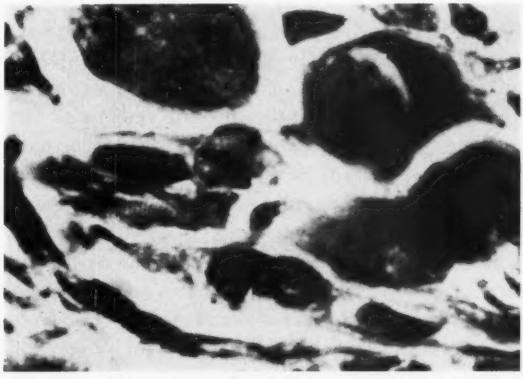

Fig. 4.—Cross striated fibers about large granular myoblasts. (Phosphotungstic acid hematoxylin; ×1,000; reduced ¼.)

involve the pleural cavities. Wolfensberger[15] first described an esophageal rhabdomyoma and the 4 previously reported cases of this rare neoplasm have recently been reviewed by DeMuth,[6] who added a fifth. Examples of intrathoracic masses of striated muscle tissue arising in teratomata are seen in the

reports of Helbing[7] and Zipkin.[17] These observations show that rhabdomyomatous tumors may arise in the region of the pleural cavities, but desipte this an extensive search of the literature has failed to reveal the report of a similar case to that just described. This is, perhaps, not a surprising finding when the unusual occurrence of solitary pleural tumors and the rarity of striated muscle neoplasms in the chest is considered.

The origin of rhabdomyosarcomas has always been a source of interest to histologists. Willis[14] says that striped muscle neoplasms arise either in immature muscle tissue or in multipotent mesenchyme, attempting by this to explain the young age distribution and unusual site of origin of many of these growths. In this case, it was unfortunate that at autopsy it was not possible to define exactly the point of origin of the tumor. However, both Wolfensberger[15] and Capell[2] have commented on the unusual pedunculation which takes place when rhabdomyomatous tumors occur beneath a surface. It seems likely that this tumor arose in either subpleural striated muscle such as diaphragm or in multipotent mesenchymal tissue situated between the lungs or the chest wall and the pleural lining cells. Subsequent pedunculation of the growth, in the same manner as that seen in these other striated muscle tumors, could have produced the final morbid anatomic picture. The intrapleural leiomyosarcoma described by Catron,[4] which resembled the present neoplasm quite closely, seemed to arise in a similar fashion. The absence of any other abnormal tissues from the tumor excluded the possibility of a teratomatous origin for the mass.

A further point of interest lies in the relatively benign character of the neoplasm. Capell and Montgomery[3] consider that those tumors which show well-differentiated myoblastic cells and which develop well-defined cross-striated fibers frequently prove to be the most malignant. The present tumor showed both these features and histologically it was considered to be a malignant neoplasm, yet, despite the fact that the mass must have been present in the patient's chest for many years, no invasion outside the tumor capsule was found at autopsy. Previously Klemperer and Rabin[9] have commented on the apparently benign nature of certain neoplasms when the mass is wholly enclosed within the pleural cavity but they offered no explanation for this phenomenon and none has since been forthcoming.

Consideration of Klemperer and Rabin's work on intrapleural tumors raises the question of the origin and nomenclature of these tumors in general. They recognized two types, a localized connective tissue group and a diffuse malignant epithelial group. They considered that the relatively benign localized connective tissue tumors arose in subpleural tissues and that subsequent growth and pedunculation of the mass produced the intrapleural site of the neoplasm. Recently Stout and Himadi[13] have suggested that all tumors which involve the pleura predominantly arise from pleural lining cells and that they be called mesotheliomas to denote such an origin. For several reasons this concept seems ill founded. It is recognized that tumors, situated within the pleura, initially will produce similar clinical pictures but they do so by virtue of their position not any special kinship between their constituent cells. The natural

histories of localized pleural tumors may vary greatly,[5] while those of the diffuse neoplasms consistently exhibit the widest possible variation.[1] Many of the reports of supposed primary diffuse pleural tumors, on critical examination, prove to be examples of metastasis and confusion of these neoplasms with the localized connective tissue types is common. At present, perfectly adequate names are available to denote connective tissue growths; to add a further all-embracing term to describe them, especially one which embraces such dubious entities as diffuse pleural tumors, serves merely to obscure knowledge of their origin. The cells lining the pleura are probably of mesenchymal origin and it is possible that they do give rise to tumors[16] but to attempt to force all pleural neoplasms, solitary or diffuse, epithelial or mesenchymal into one histogenetic classification appears highly artificial. When the tumor possesses clear evidence of its line of differentiation, as in the present case, it is also unnecessary.

SUMMARY

A case of rhabdomyosarcoma, solitary within the pleural cavity, is described. Its possible histogenesis is discussed and some features of its behavior considered.

The nomenclature of pleural tumors is briefly reviewed. It is felt that the use of the term "mesothelioma," in the classification of pleural connective tissue tumors, hinders understanding of their nature.

My thanks are due to Dr. A. W. Pound for advice and to Dr. A. D. D. Pye for permission to use hospital records.

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BRONCHOGENIC CYSTS IN INFANTS AND CHILDREN

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PULMONARY cystic disease in children is a subject which demands further clarification, inasmuch as the prognosis is so dependent on early recognition and definitive surgical therapy. Since the appearance of the first article on this subject in America by Koontz[8] in 1925, a great deal of information has been accumulated relative to this pathologic entity. However, in spite of the extensive literature, there is still much confusion concerning the nomenclature and classification of pulmonary cysts. Cooke and Blades[4] tabulated twenty-eight synonyms under which cystic disease of the lung is filed at the Armed Forces Institute of Pathology in Washington, and also collected nine others from the contemporary literature. This multiplicity in terminology is undoubtedly a reflection of how poorly the pathogenesis of this disease is understood.

The classification most generally accepted divides pulmonary cysts into congenital and acquired. This distinction, however, is based on specific histologic criteria; that is, the congenital bronchogenic variety must contain some form of respiratory epithelium in contrast to the acquired group. That this distinction has not been generally accepted is exemplified in an article by Conway,[3] who challenges the congenital origin of many of the pulmonary cysts he studied. Boyd[2] states that the so-called true congenital cyst may lose its characteristic respiratory epithelium following long-standing infection. He also notes that chronic pulmonary abscesses may have an ingrowth of bronchial type epithelium and thus simulate a true cyst.

Most authors believe that pulmonary cysts are derived from the tracheobronchial tree in one of two ways. During the fourth week of fetal life, two septa of epithelial origin divide the primitive foregut into two components. The tracheobronchial tree is derived from the anterior portion while the esophagus originates from the posterior. If cells become detached at this time, growth will continue in an abnormal fashion, resulting in the formation of bronchial or gastrogenic cysts. The other possible developmental defect occurs during the third trimester of fetal life. It is postulated that at this time a bud involving a normal or supernumerary bronchus may be pinched off and separated from the tracheobronchial tree. A pulmonary cyst may then develop in this isolated segment.

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All the pulmonary cysts reviewed in this study satisfy the criteria of the congenital bronchogenic variety, inasmuch as their histologic characteristics include some elements of the normal respiratory tract (Fig. 1). We have subdivided these cysts into two main types based upon the presenting symptom complex which they produce, namely respiratory distress or infection. These symptom complexes arise from the complications of pulmonary cysts which have previously been defined by Gross,[7] Ravitch and Hardy,[11] and Allbritten and Templeton.[1] The children in the first category entered the hospital with

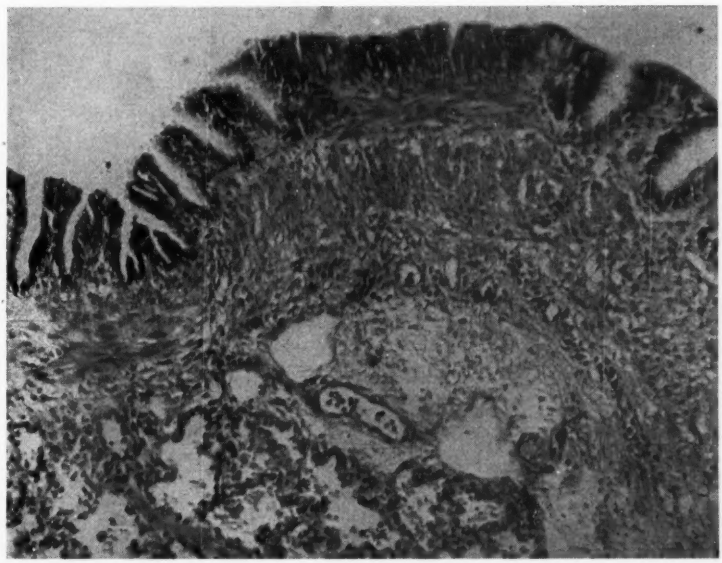

Fig. 1.—Photomicrograph of bronchogenic cyst showing columnar, ciliated epithelium, smooth muscle, and underlying alveoli. Cartilage and lymphoid tissue are not present in this specimen. (×165; reduced ⅕.)

dyspnea and frequently with cyanosis. These signs are the direct result of the space-occupying effect of the expanding lung cyst. Children in the second group are examined because of suspected pneumonia, pulmonary abscess, or recurrent bronchitis which actually are manifestations of infection occurring within a cyst. This study comprises a total of 12 patients treated surgically at this hospital during the past 15 years.

THE EXPANDING PULMONARY CYST

The development of expanding pulmonary cysts is now a fairly well-understood phenomenon. The essential mechanism for the production of this lesion is a flap-valve type of bronchial or bronchiolar obstruction. As the cyst grows larger, it gradually compresses neighboring tissue and, in an extreme degree, may occupy such a major portion of the hemithorax that the mediastinum is forced toward the opposite side. This results in the occurrence of two

further unphysiologic states: (1) compression of the contralateral lung and (2) torsion of the heart and great vessels, particularly the venae cavae, with resultant obstruction to the return flow of blood into the right side of the heart. If this process continues unchecked, death from cardiorespiratory causes may follow. Occasionally an expanding cyst may rupture into the pleural space, resulting in a tension pneumothorax, an equally serious situation.

Four of our patients were admitted either as acute or subacute respiratory emergencies. Included in this group were 2 boys and 2 girls. The youngest was 1 day of age, the oldest just under 2 months. All 4 patients entered with the similar history of rapid and progressive cyanosis and dyspnea. The pertinent physical findings were hyperresonance, decreased breath sounds, and evidence of mediastinal shift as noted by the position of the apical heart beat.

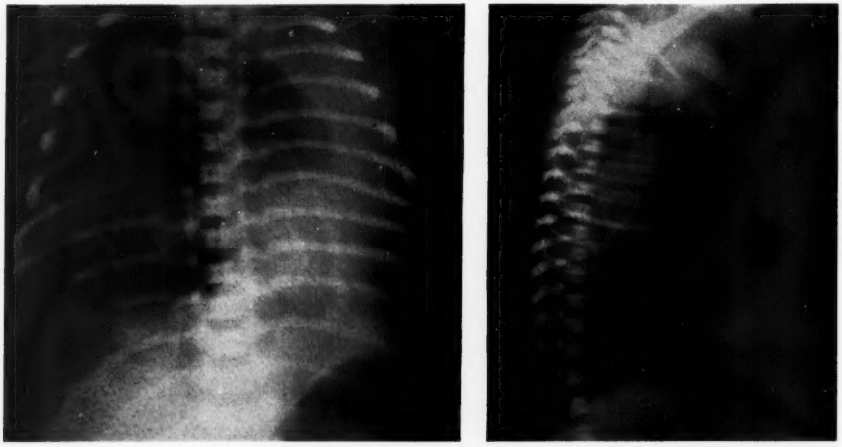

Fig. 2.—Posteroanterior and lateral roentgenograms of the chest showing a typical expanding pulmonary cyst with atelectasis and mediastinal shift.

Although the clinical picture suggested the diagnosis of an expanding cyst, this impression could be confirmed only by careful radiographic study. Roentgenograms (Fig. 2) revealed single or multiple well-circumscribed, thin-walled cystic structures which were occupying part or all of one hemithorax. The cysts partially or completely compressed the adjacent lung parenchyma and demonstrated some degree of mediastinal shift. Other confirmatory signs which indicated the expanding nature of the cyst were depression of the hemidiaphragm and widening of the interspaces on the involved side.

Two lesions which are most difficult to differentiate radiographically from the acutely expanding cyst are a tension pneumothorax and diaphragmatic hernia. It may not be possible to distinguish tension pneumothorax from pulmonary cyst before thoracotomy; however, less harm is done by surgical exploration in this situation than by temporizing with repeated thoracenteses or intercostal drainage. A real effort should be made to differentiate diaphragmatic herniae from pulmonary cysts inasmuch as the former are best corrected in infants

through an abdominal approach, whereas the latter can only be attacked by thoracotomy. When the diagnosis is in doubt, the best radiologic technique to prove the presence of a diaphragmatic hernia is by contrast study of the gastrointestinal tract.

The treatment of our 4 cases of expanding lung cysts fairly well exemplifies the changing attitude to this problem in the past decade. Before the advent of the newer techniques of anesthesia and the concomitant advances in thoracic surgery, no attempt was made to attack this emergency directly. Only supportive therapy, consisting of oxygen and needle aspiration of the cyst, was attempted and the resulting mortality was unduly high. In recent years, due to the better understanding of the physiologic problems associated with pulmonary resection, the mortality has been greatly reduced.

Three patients had resections (one pneumonectomy and two lobectomies) with uneventful postoperative courses. The fourth died as a result of an error in diagnosis. This death occurred in an infant who was thought to have a diaphragmatic hernia by roentgenography and consequently underwent laparotomy. Before thoracotomy could be performed, the child suddenly died. This case exemplifies the importance of a correct preoperative diagnosis when multiple pulmonary cysts are present.

The pathology of the resected specimens is summarized in Table I. On gross examination the specimens presented a fairly uniform picture. Single or multiple cysts could be seen lying within the lung parenchyma with surrounding atelectasis. On cross section, they had a smooth, pale, glistening surface with a glary type of mucus on the wall. Some were crossed by fine trabeculae, whereas others were multiloculated. A bronchial communication was usually demonstrable; inflammation was not prominent.

TABLE I. EXPANDING LUNG CYSTS

CASE NUMBER	LOCATION	NUMBER OF CYSTS	HISTOLOGY OF CYST WALL
1	RML	One large, numerous small	1. Columnar epithelium 2. Smooth muscle
2	RLL	One large, numerous small	1. Columnar epithelium 2. Smooth muscle
3	RML	Single large	1. Columnar epithelium 2. Smooth muscle
4	LLL	Single large	1. Columnar epithelium 2. Smooth muscle

In the management of the child with an expanding pulmonary cyst, the following points should be stressed. It is essential that the preoperative period occupy as short an interval as possible and be concerned only with making the diagnosis. During the time necessary for carrying out diagnostic procedures, supportive therapy, mainly oxygen, should be given. Needle aspiration of a tension cyst plays no part in the definitive treatment of this condition, as shown by Maier and Haight.[9] Thoracentesis should be reserved only for the occasional case which requires emergency decompression until surgery can be performed. Once the diagnosis is made, immediate thoracotomy is mandatory with removal of the expanding cyst. The type of excision may vary from

pneumonectomy to segmental resection. The lung should be completely palpated to determine the presence of additional cysts. If the lesions are in the lower lobe, a sequestrated lung should be suspected and a careful search made for the aberrant arterial supply.

THE SECONDARILY INFECTED CYST

The secondarily infected pulmonary cyst occurs as a result of bacterial invasion of a previously sterile cyst. These cysts may be asymptomatic for years, and then suddenly make their presence known in an explosive fashion as an acute inflammatory process. According to Croswell and King,[5] they are originally fluid-filled cavities without bronchial communication and, as such, remain sterile. However, when perforation into a bronchus occurs, bacteria may enter, resulting in secondary infection. Once inflammation has begun, it is difficult to control, since the cavities seldom have dependent drainage. During the acute stage of the infection, there is usually extension of the process beyond the confines of the cysts, resulting in pneumonitis and, at times, empyema. However, on adequate antibiotic therapy, the inflammation may subside, but a nidus remains for later recurrent episodes. Some cysts become a source of chronic pulmonary suppuration with bronchiectasis, and eventually pulmonary insufficiency develops.

Eight cases of secondarily infected pulmonary cysts were evaluated in our study. These children were in an older age group than those with the expanding type of lung cyst. The youngest was 6 months and the oldest 9 years. Of these, 5 were male and 3 female.

These children with the secondarily infected cysts have a classical clinical history of intermittent respiratory infections with asymptomatic intervals. The first attacks are usually interpreted as bronchitis or pneumonia. Following several such episodes, however, roentgenograms of the chest usually reveal the correct diagnosis. The most common symptoms and signs in our patients during the acute stage of their disease were those of pulmonary suppuration, namely, fever, dyspnea, cough, and purulent sputum. Physical findings were not remarkable during the periods of remission, but during the acute attacks they were suggestive of pneumonitis. Laboratory studies of a routine nature only confirmed the presence of infection, and frequently demonstrated a previously unsuspected anemia.

The radiographic picture (Fig. 3) seen in the secondarily infected cyst differed from the expanding cyst in that there was little evidence of compression of surrounding structures. The cysts appeared extremely round, smoothly outlined and thin-walled with varying degrees of pneumonitis and frequent evidence of fluid levels. The latter, however, usually presented with a thicker wall and a great deal more surrounding pneumonitis. A second type of lesion to be considered in the differential diagnosis is the staphylococcal pneumatocele. However, in this disease, pneumonia is the predominant feature. Furthermore, the natural course of this lesion is spontaneous regression over a period of months, which never occurs in the true pulmonary cyst.

The differential diagnosis between the secondarily infected cyst and the previously mentioned pulmonary infections is often extremely difficult and rests in an accurate evaluation of the history, clinical course, and serial roentgenograms. Infected pulmonary cysts characteristically present as acute respiratory infections with relatively asymptomatic intervening periods, whereas, lung abscess entirely resolves without exacerbations or continues as a chronic suppurative process. Roentgenograms may at times be of value in distinguishing these two conditions. This is of some importance, since in recent years excellent results have been obtained in the treatment of the acute abscess with antibiotics.

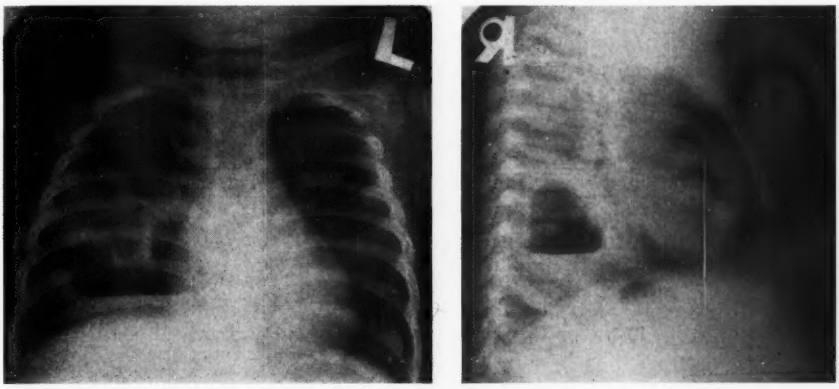

Fig. 3.—Posteroanterior and lateral roentgenograms of the chest showing the well-circumscribed appearance of an infected pulmonary cyst with a fluid level and minimal surrounding pneumonitis.

In contradistinction, infection in a congenital cyst can only be controlled by chemotherapy, but never cured, inasmuch as the underlying cause still remains. Consequently, surgery is the treatment of choice in the latter while conservative means should be employed in pulmonary abscess unless the course of the disease becomes chronic.

The necessity of differentiating the staphylococcal pneumatocele from pulmonary cysts is imperative as shown by the observations of Potts and Riker[10] and Fisher and Swenson,[6] who emphasize that staphylococcal pneumatoceles usually regress spontaneously. Thus, in any case in which the radiographic interpretation is suggestive of lobar ectasia, the history, clinical course, and serial roentgenograms over a period of several months should be reviewed before any thought is given to excisional surgery.

The preoperative preparation in children with secondarily infected pulmonary cysts can be divided into two phases, (1) therapeutic and (2) diagnostic. The therapeutic considerations consist mainly of antibiotics to control the infection during the acute stage. Once this has been accomplished, therapy should be directed toward decreasing the tracheobronchial secretions to a minimum by chemotherapy, postural drainage, and bronchoscopic aspiration, if necessary.

Blood replacement prior to surgery may be necessary in many of these children with prolonged or recurrent infections. Diagnostic and therapeutic bronchoscopy should always be performed preoperatively to rule out the presence of a foreign body or cystic bronchiectasis. If bronchiectasis is suspected, bronchography should be undertaken to confirm the diagnosis and delineate the extent of the disease.

In the eight infected pulmonary cysts studied, three were treated by pneumonectomy, four by lobectomy and, one of the earliest cases, with marsupialization and subsequent thoracoplasty. There were two deaths in this group; 1 patient died shortly following pneumonectomy after several episodes of cardiac arrest during the procedure and the other patient died on the operating table during right middle lobectomy. Both deaths were attributable to massive flooding of the contralateral lung by infected material from the cysts, and would be preventable deaths today with our present surgical knowledge.

TABLE II. SECONDARILY INFECTED PULMONARY CYSTS

CASE NUMBER	LOCATION	NUMBER OF CYSTS	HISTOLOGY OF CYST WALL
1	LLL	Multiple small	1. Columnar epithelium
2	RML	One large, multiple small	1. Columnar epithelium 2. Smooth muscle 3. Cartilage 4. Chronic inflammatory reaction
3	RLL	One large	1. Columnar epithelium 2. Smooth muscle 3. Chronic inflammatory reaction
4	LUL and LLL	Multiple small	1. Columnar epithelium 2. Smooth muscle 3. Chronic inflammatory reaction
5	RLL	One large	1. Islands of columnar epithelium 2. Chronic inflammatory reaction
6	RLL	Two large	1. Columnar epithelium 2. Cartilage 3. Smooth muscle 4. Chronic inflammatory reaction
7	RLL	One large	1. Columnar epithelium 2. Smooth muscle 3. Chronic inflammatory reaction
8	RLL	Multiple small	1. Columnar epithelium 2. Smooth muscle 3. Chronic inflammatory reaction

These 2 cases emphasize the importance of the proper preoperative preparation aimed at controlling the quantity of secretions in these cysts by antibiotics, postural drainage, and bronchoscopy. The other children in this group had uneventful postoperative courses and have remained well to date.

The pathologic findings of these surgical specimens are summarized in Table II. As can be seen, there is a definite predilection for these cysts to occupy the lower lobes. Grossly, they appear similar to the expanding pulmonary cyst except that evidence of infection supplants the signs of compression

of neighboring pulmonary tissue. On cross section, the bronchial type mucosa has a glistening appearance. The cavity itself usually contains purulent exudate and there is evidence of pneumonitis in the surrounding parenchyma.

Since there is seldom the fulminating life-endangering process occurring in the secondarily infected cyst, as there is with the expanding type, a more leisurely attitude may and should be taken in the handling of this problem. It is essential that the proper length of time be utilized to prepare these children adequately for surgery and also to insure the correct diagnosis. This frequently demands time-consuming observations of the clinical course and complete preoperative studies. Once the above requirements have been fulfilled, the indicated pulmonary resection should be performed. The postoperative care is essentially the same as in any child who has undergone thoracotomy.

DISCUSSION

Only 20 years ago Schenck[12] stated that tension-type pulmonary cysts were attended by a grave prognosis in infants and children. Acute inflammatory lesions of the lung likewise carried a high mortality during that period. The gloomy outlook has now changed and the prognosis of this disease is excellent, as exemplified by the survival of 75 per cent of the patients treated at this hospital by resection. This improvement can be traced, in part, to refinements in surgical technique and care, but more important to the earlier suspicion and consequent diagnosis of this entity. Early recognition of this relatively rare but serious disease can only be accomplished by constantly being alert to its possible existence. Every infant with respiratory distress should have a roentgenogram of the chest to rule out the presence of an expanding pulmonary cyst. Recurrent bouts of pulmonary infection in childhood should be viewed with suspicion and similar appropriate diagnostic studies undertaken. Once the diagnosis is established or strongly suspected, excisional surgery is the procedure of choice. If this plan of therapy is followed, children with complicated cystic disease of the lung will come to definitive treatment earlier and the morbidity and mortality will be concurrently lowered.

SUMMARY

1. Congenital pulmonary cysts may be divided into two major groups based upon either of two presenting symptom complexes associated with expansion or infection.

2. Expanding pulmonary cysts are usually found in the newborn period; since they may run a rapidly fulminating course, prompt diagnosis and surgery are mandatory.

3. Infected pulmonary cysts occur in an older age group and usually require a more protracted period for accurate diagnosis and adequate preoperative preparation.

4. The treatment of choice in both types is by resection of the involved pulmonary parenchyma.

5. An excellent prognosis can be predicted in this disease with early diagnosis and proper surgical management.

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GIANT INTRATHORACIC LYMPH NODES

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BENIGN hyperplasia, producing giant mediastinal lymph nodes,[1] adds another consideration to the difficult preoperative evaluation of mediastinal masses and is particularly important because, both grossly and histologically, it may be mistaken for a thymic tumor. Because of the possible relationship of thymic function and thymic tumors to the crippling disease, myasthenia gravis, such an error may be harmful to a patient and might confuse the further study and classification of this problem. Furthermore, prognosis for the lesions may vary.

Castleman and associates[2] reported a case of benign hyperplasia of mediastinal lymph nodes, and Castleman, Iverson, and Menendez[3] collected 13 cases from an extensive study of thymic tumors. Etiologically, they favor chronic inflammation rather than new growth of thymic origin. The initial diagnoses of ectopic thymic tumor in 1 case[4] and primary subpleural thymoma in another[5] were changed to benign hyperplasia of lymph nodes upon review.

In most cases, a routine or survey film of the chest discloses an asymptomatic mass protruding from either lung hilum or occupying the anterior mediastinum. Surgical excision is usually simple and not followed by recurrence or complication. The gross specimen usually presents a firm, well-encapsulated mass with a finely granular cut surface. Microscopically, there is marked lymphoid hyperplasia with capillary proliferation in various stages. Often there are highly reactive germinal centers with hyalinization and lymphocytic cuffing. These may closely resemble the Hassall's corpuscles of the thymus and thereby may lead to an erroneous diagnosis.

CASE REPORTS

CASE 1*.—Chinese male, 23 years old. An asymptomatic left hilar mass gradually enlarged as demonstrated in chest films over a 3-year period (Fig. 1). All preoperative examinations, including sputum studies for bacilli and cells, tomograms, bronchoscopy, and angiocardiography failed to clarify the diagnosis. In September, 1954, a left thoracotomy demonstrated an egg-sized mass in the interlobar fissure of the left hilum, with no invasion into the surrounding lung parenchyma. The mass shelled out easily as it was well encapsulated.

From the Department of Surgery, Leahi Hospital, Honolulu, Hawaii.
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*Case from the service of Dr. Paul W. Gebauer.

Frozen section study was reported as showing a "lymph node." The pathologic description by Dr. R. B. Chappell* was as follows: "A thin and completely encapsulated solid structure of the general appearance of a markedly enlarged lymph node. It is 4.1 cm. in greatest diameter and ovoid (almost spherical) in shape. Sections show a homogenous parenchyma of essentially normal consistency and a fine granularity representing, no doubt, the follicles of the node. Histological sections [Fig. 2] show a lymph node in which the only real, essential abnormality is its giant size. It has a preserved follicular pattern, and many of the germinal centers show an incidental nonsignificant finding consisting of hyaline and epithelioid cell masses. These late changes are probably the result of long-standing inflammation and/or hyperplasia. No malignant features. Diagnosis: Hyperplasia and hypertrophy of lymph node, benigh."

Recent follow-up studies 7 years after the first abnormal chest film revealed no recurrence.

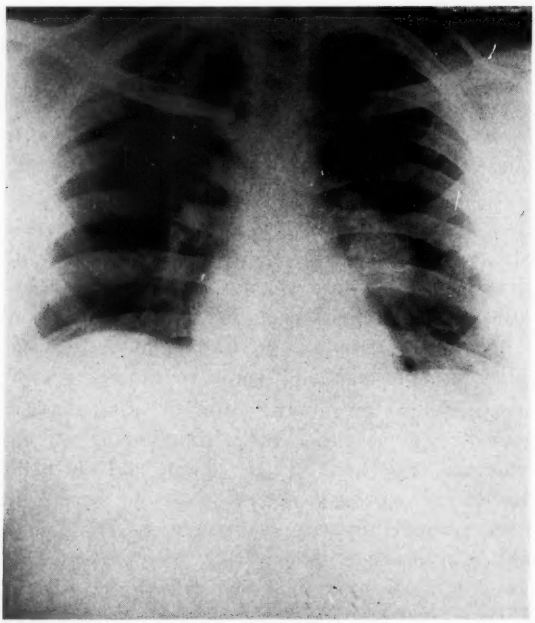
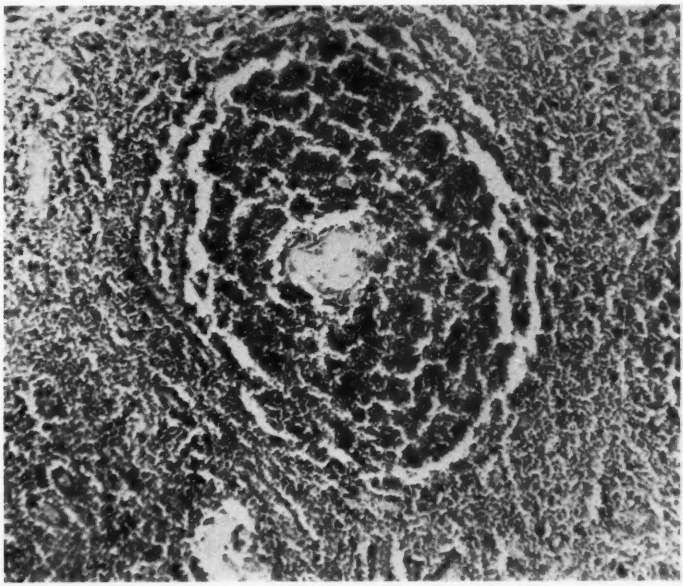


Fig. 1.—A well-circumscribed left hilar mass reported to have increased in size over a 3-year period is shown in the posterior-anterior chest film.

CASE 2†.—Japanese male, 13 years old. A persistent nonproductive cough for 6 weeks followed an upper respiratory infection. A roentgenogram of the chest (Fig. 3) revealed a mass in the right hilum. This was regarded as a bronchogenic cyst and, at operation in July, 1955, a solid mass "resembling a group of lymph nodes" was found in the lower right hilum. The mass easily shelled out. There was no evidence of local invasion. The frozen section diagnosis was "benign thymoma." The gross specimen consisted of a pink lobulated mass weighing 32 Gm. and measuring 5 by 4 by 3 cm. The cut surface showed uniform, meaty, soft, pink tissue throughout. Dr. W. Harold Civin‡ described the microscopic findings as follows: "Histological examination [Fig. 4] of the tissue from the mediastinum reveals evidence of a hyalinized fibrous band separating masses composed

*Pathologist, St. Francis Hospital, Honolulu, Hawaii.
†Case from the files of Dr. R. G. Johnston.
‡Pathologist, The Queen's Hospital, Honolulu, Hawaii.



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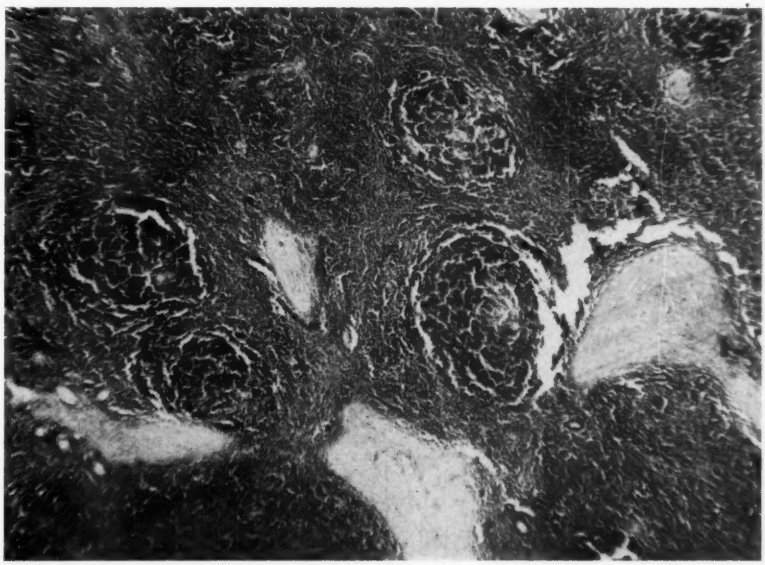

B.

Fig. 2.—*A*, Histologic examination sections show hyaline and epithelioid cell masses within the germinal centers. *B*, Under low power the typical lymph node architecture is seen.

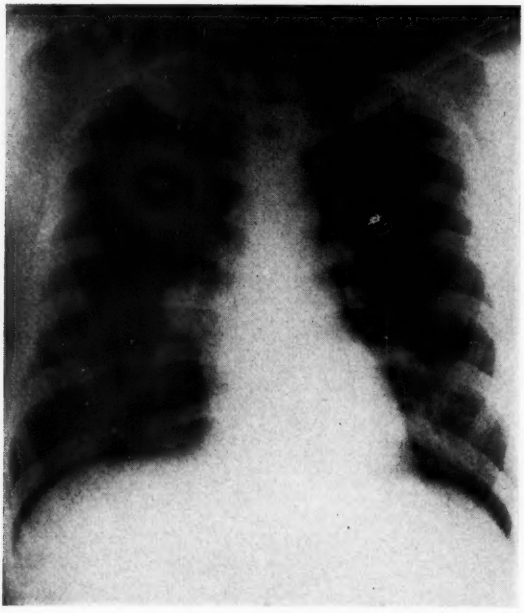

Fig. 3.—A small, circumscribed right hilar mass is seen in the posterior-anterior chest film.

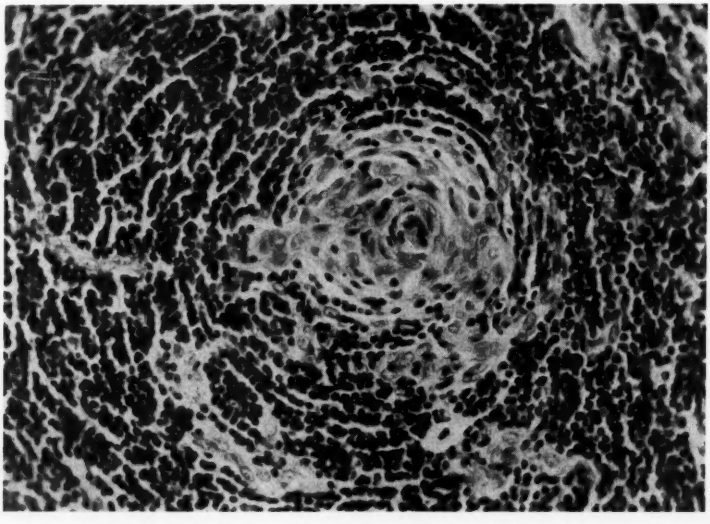

Fig. 4.—Histologic examination section shows the so-called "pseudo-Hassall corpuscles," consisting of the epithelioid cell masses and hyaline characteristic of hyperplastic lymph nodes.

of thymocytes and reticular cells; the latter tend to form pseudo-Hassall corpuscles. There is some tendency toward rosette formation of thymocytes. The picture is that of thymoma and is benign. There are hyaline bands between the lymphoid cells. This is rather characteristic of this particular lesion. Diagnosis: Benign thymoma."

In August, 1957, after reviewing all thymomas removed at Queen's Hospital, the same pathologist reported: "Review of this slide in view of later knowledge leads me to class this an a localized hyperplasia of a mediastinal lymph node and still benign. The previously called thymocytes are lymphocytes, undoubtedly. This case has been removed from the thymoma classification. Amended diagnosis: Localized hyperplasia of a mediastinal lymph node."

CASE 3*.—Filipino male, 49 years of age. A yearly survey film of the chest revealed a mass in the anterior mediastinum, which was asymptomatic and had been present for about 1 year. Preoperative sputum studies, angiocardiography, and bronchoscopy revealed no abnormality. The preoperative diagnosis was thymoma or dermoid. In August, 1957, a left thoracotomy revealed a 6 cm. mass lying beneath the mediastinal pleura, superior to the pulmonary artery and anterolateral to the ascending thoracic aorta. The mass easily shelled out, with no evidence of local invasion. Frozen section studies revealed either "lymphoma or thymoma."

The pathologic report was as follows: "The specimen consists of a mass, well encapsulated, ovoid in shape, measuring 8 cm. in its greatest dimension and 6 cm. in its least dimension. Weight 100 Gm. Cut section reveals a lobular pattern and an attempt at lobular formation. Areas of subcapsular hemorrhage are seen. Histological examination reveals a markedly enlarged lymph node, which shows severe hyperplasia of both lymphoid and reticular elements, but with good preservation of the architecture. Normal germinal centers, small vessels, and peripheral sinuses are noted. The cells are well differentiated and uniform. No thymic elements can be seen. The picture is that of an unusually marked but benign hyperplasia, presumably of inflammatory origin. Diagnosis: Mediastinum, benign hyperplasia of lymph node."

However, after multiple sections and further studies by numerous pathologists, in a supplementary report the diagnosis was changed to: "Mediastinal tumor, thymoma, most likely benign."

SUMMARY

These cases illustrate the salient clinical and pathologic features of giant intrathoracic lymph nodes and demonstrate the necessity of thoracotomy for diagnosis. The second case suggests that histologic review of thymomas by pathologists might disclose more instances of benign hyperplasia and help clarify the difficulties existing in their diagnosis and identification. Case No. 3 is a case in point which illustrates the difficulty in the differentiation of benign hyperplasia of lymph nodes from thymic tumors. All of the cases indicate the importance of careful study of a given lesion for accuracy so that behavior, treatment results, and prognosis may perhaps be clarified.

The writer wishes to thank Dr. P. W. Gebauer for his assistance in this paper, Dr. I. L. Tilden for the photographs of the histologic sections, and Mr. I. Ota for the chest pictures.

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CONGESTIVE ATELECTASIS: A FATAL POSTOPERATIVE COMPLICATION

Hurst B. Hatch, Jr., M.D., and G. M. Carrera, M.D., New Orleans, La.

SINCE the original description, by Jenkins and associates,[1] of postoperative congestive atelectasis, this complication has received little attention in the medical literature. It probably occurs more often than this scarcity of reports would indicate. We became interested in this condition about 4 years ago, when we encountered our first patient with this complication. The clinical picture presented by this patient was one of sudden onset of severe dyspnea, deep cyanosis, fever, labored expiratory breathing, tachypnea, tachycardia, and hypotension, aggravated by infusion of fluid, particularly blood. There were no pathognomonic roentgenographic signs. The condition was characteristically resistant to all forms of therapy directed toward pulmonocardiac failure. Since then we have seen 6 additional patients in whom this condition developed after a thoracic surgical procedure. It is the purpose of this report to call attention to this entity by discussing it in the light of our experience with these 7 cases.

Congestive atelectasis is a complication of parenteral fluid therapy. These complications may be grouped into four major types: (1) pyrogenic reactions, (2) volume variations, combined with changes in osmolar concentration, (3) compositional disturbances, and (4) "speed" reactions. Predominantly, congestive atelectasis is a complication of the fourth type.

This entity was described by Bert[2] as attending rapid decompression. The description of the gross and microscopic findings of the lung in rapid decompression, as reported by Fegler and Banister,[3] is almost identical with those described by Muirhead,[4] of patients receiving large amounts of intravenous fluids rapidly. Grossly, the lungs of congestive atelectasis are heavy, and macroscopically the condition suggests gross hemorrhage. The areas affected are sharply limited; they are liver-like, uniform, and dark red. The affected portions seem to be commoner around the hilum and on the dorsal surfaces of the lungs. Microscopically, intense capillary congestion, intra-alveolar hemorrhage, minimal pulmonary edema, and incomplete expansion of pulmonary tissue are seen.

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In addition to the excessive and rapid infusion of fluid, probably other factors contribute to development of this complication. Physical injuries, such as surgical procedures, burns, blast injuries, anaphylaxoid shock, or pre-existing shock from some other cause, are the most common. However, none of these alone would necessarily be the cause of death.

The onset of congestive atelectasis is sudden. The dyspnea is severe and indicative clinically of an expiratory obstructive type of breathing. The cyanosis is deep and resistant to all forms of therapy. The tachycardia and hypotension are pronounced and progressive.

The physical signs are those usually found in a patient with acute left ventricular failure. However, in some cases the classical findings of acute left ventricular failure, particularly multiple moist râles throughout the pulmonary field, are absent.

Probably one of the more frequent surgical complications is obstructive collapse of pulmonary tissue. This complication should not be confused with congestive atelectasis, as the two conditions differ, not only in their clinical manifestations but also in their response to therapy. In Table I comparison of the four major physical findings is shown.

TABLE I. DIFFERENTIATION BETWEEN OBSTRUCTIVE AND CONGESTIVE ATELECTASIS

PHYSICAL FINDING	OBSTRUCTIVE	CONGESTIVE
Cyanosis	Light	Deep
Response to oxygen	Good	Poor
Mediastinal shift	Present	Absent
Bronchoscopy	Helpful	Not helpful

Characteristically, all known treatment has been futile. Administration of digitalis and oxygen, and performance of phlebotomy and bronchoscopic aspiration have been to no avail. Inhalation of carbon dioxide, frequent turning of the patient, administration of steroids, and coughing have had no beneficial effect. Despite all known resuscitative measures, the fully developed syndrome is always fatal.

In our 7 patients there were two common factors: (1) rapid infusion of large amounts of fluid during and after a surgical procedure, and (2) a thoracic operation. Six of our patients were men and 1 was a woman. Their ages ranged from 44 to 65 years. Since the pattern in all 7 cases was similar, only one will be reported in detail.

REPORT OF CASE

A white man, aged 44 years, was admitted to Ochsner Foundation Hospital in July, 1956, because of hemoptysis of 1 week's duration. At the age of 11 years, after tonsillectomy, an abscess developed in the right upper lobe of the lung. Empyema subsequently developed and this was drained.

No abnormalities were detected on physical examination the day of admission. Results of routine laboratory studies were within normal limits. In roentgenograms of the chest, retraction of the transverse fissure superiorly, with a radiolucency, 2 cm. in diameter, and some haziness surrounding this area of radiolucency in the anterior segment

were noted. In planograms, definite cavitation was seen in the anterior segment of the upper lobe. On bronchography, an outline of the cavity in the anterior segment was seen with cystic bronchiectasis in the right upper lobe.

Right upper lobectomy was done. Within 4 hours the patient received 2,500 c.c. of blood and 2,000 c.c. of glucose in water. Soon after his return from the operating room he had an acute hypotensive episode accompanied by progressive dyspnea and severe cyanosis with pronounced tachycardia. Because of the severe hypotension, within 17 hours the patient received 9,000 c.c. of whole blood, of which 1,500 c.c. had been transfused at a rapid rate. Despite all efforts to reverse the hypotension, the patient died 24 hours after operation.

The gross and microscopic pictures (Fig. 1) were typical of congestive atelectasis.

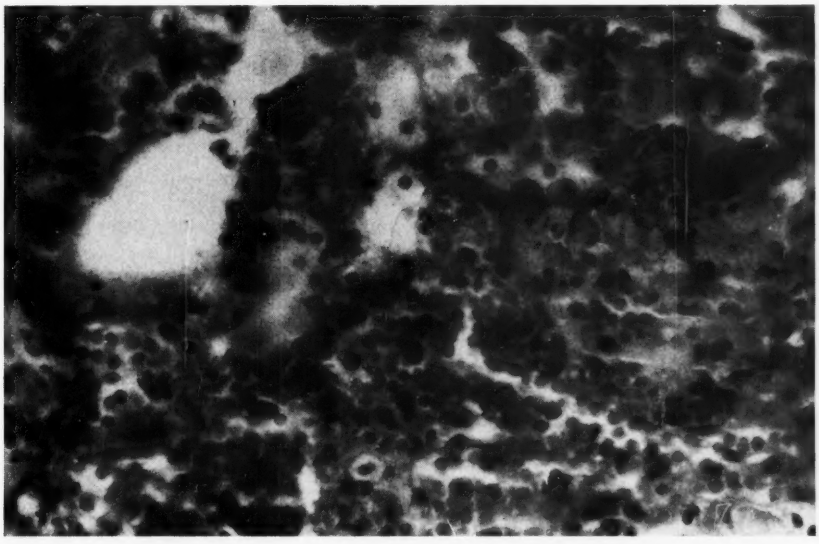

Fig. 1.—Case 1. Lung. There is extensive congestion of the capillaries in the alveolar septa, with some extravasation of red blood cells. The alveolar spaces show compression by the congested, edematous parenchyma, and contain coagulated protein and red cells. (Hematoxylin and eosin, ×430; reduced ¼.)

DISCUSSION

The experimental studies of this condition by Moyer[5] are fascinating. He reproduced, in animals, every manifestation of congestive atelectasis noted in human beings except the severe hypotension. He concluded that his experimental findings did not completely clarify the etiology of non-decompression atelectasis. However, his studies have contributed to some understanding of the phenomenon. Several facts are apparent from his work. First, the volume of blood in the lungs increases rapidly after hemorrhage or asphyxia and, second, stiffening of the lung begins soon after rapid parenteral injection of solutions, even in an animal which is not in shock. He noted that the stiffness became pronounced as the left atrial pressure fell. This rules out left ventricular failure as a universal cause of acute development of pulmonary stiffness postoperatively.

Physiologically, an initial rise is followed by a drop in the pulmonary arterial pressure. Then, a fall in systemic pressure occurs and is progressive; the residual air decreases and the respiratory rate increases. As evidence of stiffness or loss of compliance in the lungs, the tidal volume is decreased or is unchanged even as the inspiratory intrapleural pressure becomes more negative than the expiratory intrapleural pressure becomes more positive, thus increasing the work of breathing. It is apparent that the cyanosis represents continuance of pulmonary blood flow through the capillary area in the presence of airless alveoli, a definite abnormality of ventilation and perfusion.

Since our experience with this syndrome, we have been extremely cautious in the postoperative administration of parenteral fluids. Obviously, the best therapy for this fatal syndrome is preventive. In all patients in whom hypotension develops postoperatively, it is imperative to determine the cause, if possible, and treat it physiologically rather than to rely on infusion of fluids to raise the blood pressure to an accepted clinical level. Since low blood pressure is a sign of disease, we are actually treating the effect rather than the cause of the disease. Every hypotensive episode occurring during or immediately after operation is not necessarily due to loss of blood.

SUMMARY

Congestive atelectasis, or acute pulmonary congestion, a little known postoperative complication, is discussed in the light of experience with 7 cases. We believe that this complication is preventable. It is hoped that this report will stimulate the clinician to determine the cause of shock and thus treat it physiologically rather than mechanically.

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INTRATHORACIC MENINGOCELE

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INTRATHORACIC MENINGOCELE was first described in the literature by Pohl in 1933. The next recorded surgical attack was carried out by Ameuille and associates in 1940. The meningocele was excised, and the resulting defect packed. A spinal fluid fistula and an empyema combined to produce a fatal issue.

Isolated case reports and reviews of the literature have appeared from time to time, the latest being that of Raison. According to our own computation, there have been 27 verified cases reported in the world literature to date, making the patients reported in this communication the twenty-eighth and twenty-ninth, respectively (Table I). Data are incomplete in some case reports. These include the case of Freedlander, the case seen incidentally at necropsy by Kay, the case of Head (quoted by Baker and Curtis, and Raison), 1 case of Byron and associates, and that of Ottani (quoted by Raison).

Surgical correction has been undertaken in 14 patients. Four have died from causes related directly to surgical intervention. The first 2 cases reported were surgical fatalities. Since 1948, however, there have been 12 operations with 2 deaths (Table II).

The etiology of intrathoracic meningocele is, of course, speculative, and numerous explanatory theories have been postulated. It could represent the result of a primary defect in the posterior neural arch. Abnormal extension of the dura around the emerging spinal nerves is another possibility. Localized disease of the dura might conceivably render it too weak to resist pressure of the subarachnoid space, resulting in saclike dilatations of the intraspinal meninges through an enlarged intervertebral foramen. The frequent association of this entity with neurofibromatosis is striking, and may be significant. Fifteen of the 27 cases reported in the literature have been associated with neurofibromatosis. Some authors, in fact, consider intrathoracic meningocele simply to be one manifestation of von Recklinghausen's disease. Neurofibromatosis is known to be characterized by developmental disturbances in neural tissue and occasionally by bony changes, including scoliosis and

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TABLE I

AUTHOR	SEX	AGE	CUTANEOUS NEUROFIBROMATOSIS	SITE OF ORIGIN	OPERATION	REMARKS
Pohl (1933)	F	47	Yes	D3-4	Yes	Died of empyema
Schuller and Uiberall (1938)	M	56	Yes	D7-8	No	Died following resection of posterior roots 6, 7 and 8
Ameuille et al. (1940)	F	48	No	D8-9	Yes	Died of empyema and spinal fluid fistula
Freedlander (1946)	F	?	Yes	?	Yes	?
Singpiel et al. (1947)	M	21	No	D1-2	No	
Mendelsohn and Kay (1948)	F	40	Yes	D7-8	Yes	Recovery
Kay	One case seen incidentally at autopsy					
Welch et al. (1948)	F	41	Yes	D6-7	Yes	Recovery
Byron et al. (1948)	F	36	No?	D2-3	Yes	Recovery
	M	61	Yes	C7-D1	Yes	Died from hemorrhage
	M	21	No	D1-2	No	
Head (1948)	M	43	No	?	No	
Cross et al. (1949)	M	22	Yes	D8-9	No	
Ciaglia (1952)	F	31	Yes	C7-D1	Yes	Recovery
Rubin and Stratemeier (1952)	?	7 wks.	No	D4-5	No	Died
Baker and Curtis (1953)	F	40	Yes	D8-9	Yes	Recovery
	M	63	No	D9-10	Yes	Recovery from operation, but died 7 months later of Paget's disease
Sears et al. (1953)	F	29	No	D2-3	Yes	Recovery
Kessel (1951)	F	23	Yes	D5-6	Yes	Recovery
	F	8	Yes	D7-8	Yes	Operation done to relieve paraplegia; resection of vertebral body only
Turunen (1953)	M	33	Yes	D5-6	Yes	Recovery
Ottani (1951)	Incomplete data—proved by aspiration					
Hackenseller (1953)	F	45	Yes	D8-9	No	Died following irradiation therapy
Portigliatti-Barbos (1953)	F	60	Yes	D6-7	No	Died following coughing spell
Gernez-Rieux and Lepaul (1954)	M	59	No	D11-12	No	
Raison (1956)	M	39	No	D8-9	Yes	Recovery
	F	46	Yes	D3-4	Yes	Died from cerebrospinal leakage

TABLE II. SURGICAL TREATMENT OF MENINGOCELE

AUTHOR	OPERATION	RESULTS
Pohl (1933)	Myeloplasty	Died of empyema
Ameuille et al. (1938)	Packing	Died of empyema and spinal fluid fistula
Mendelsohn and Kay (1949)	Myeloplasty	Recovery with satisfactory result
Welch et al. (1948)	Myeloplasty	Recovery with satisfactory result
Byron et al. (1949)	Myeloplasty	Recovery with satisfactory result
	Myeloplasty	Died from hemorrhage during operation
Kessel (1951)	Myeloplasty	Recovery with satisfactory result
Ciaglia (1952)	Myeloplasty	Recovery with satisfactory result
Baker and Curtis (1953)	Myeloplasty	Recovery with satisfactory result
	Myeloplasty	Recovery from operation but died 7 months later of Paget's disease
Sears et al. (1953)	Myeloplasty	Recovery complicated by spinal fluid fistula and meningitis; ultimate recovery with good result
Turunen (1953)	Myeloplasty	Recovery with satisfactory result
Raison (1956)	Myeloplasty	Recovery with satisfactory result
	Myeloplasty	Died from spinal fluid fistula
Hilton and McCarthy	Myeloplasty	Recovery with satisfactory result

growth abnormalities. The fact remains, however, that intrathoracic meningocele can and does exist independently of neurofibromatosis. A relationship undoubtedly exists, but its causal nature is open to question.

Deformities of the thoracic spine ordinarily accompany an intrathoracic meningocele. In fact, 23 of the 27 cases reported manifest vertebral abnormalities adjacent to the tumor. For the most part, they are consistent with pressure effects exerted by the cystic mass. Vertebral changes commonly observed include kyphosis, scoliosis, kyphoscoliosis, erosion or destruction of vertebral pedicles. Occasionally, the ribs contiguous to the meningocele are spread, resulting in subluxation of the costovertebral articulation. The intervertebral discs are unaffected.

Neurofibromatosis is seldom associated with posterior mediastinal neurogenic tumors. Kent and Blades ascertained from a large series that this combination is so infrequent that it is statistically of no significance.

There is no constant or characteristic symptomatology related to intrathoracic meningocele. A survey of the literature indicates that some individuals seek medical advice for a variety of reasons, including pain in the chest, shoulder, and thoracic spine; persistent unexplained coughing; and intractable headache. Others become apprehensive about a progressive spinal deformity.

Many are asymptomatic, a posterior mediastinal tumor having been divulged by a roentgenogram of the chest taken during the course of a complete physical examination.

Since intrathoracic meningocele is a rare entity, it is seldom suspected and, therefore, almost never diagnosed preoperatively. A posterior mediastinal tumor discovered in a patient with von Recklinghausen's disease should, however, lead one to suspect it. If, in addition, there are associated skeletal changes one may confidently predict it. Only myelography demonstrating a passage of the contrast medium from the subarachnoid space into the mass can confirm it.

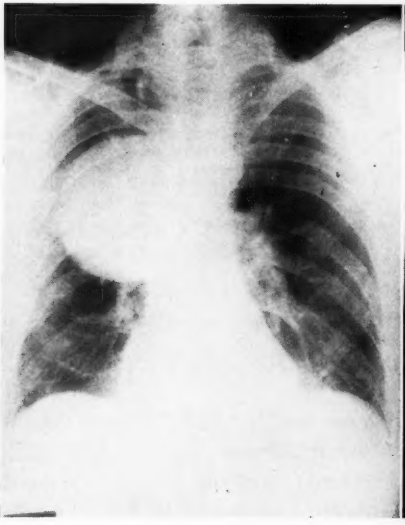

Fig. 1.

Fig. 2.

Fig. 1.—Anteroposterior roentgenogram of the chest in Case 1 demonstrating a mediastinal mass projecting into the right hemithorax.

Fig. 2.—Lateral roentgenogram of the chest in Case 1 demonstrating the posterior situation of the mediastinal mass.

Because intrathoracic meningocele represents a progressively expanding lesion, subject to rupture and prone to pressure effects upon surrounding structures, the treatment of choice is surgical repair of the defect. This is done through a posterolateral thoracotomy incision. The dural sac is dissected free from the pleura and adjacent structures until the dilated intervertebral foramen is encountered. It is then opened and trimmed, taking care to leave enough dura for the repair. The dura and its overlying pleura are then imbricated separately so as to give a firm and water-tight closure. It is probably prudent to place an intercostal catheter in the pleural space for the application of continuous suction for 24 hours postoperatively. In this way, complete re-expansion of the lung is assured and the dural repair reinforced.

Postoperative mortality and complications are derived from infection (meningitis and empyema) and spinal fluid leakage with fistula formation. Meticulous aseptic technique should forestall the development of the former and careful closure of the defect of the latter. If spinal fluid leakage does occur, the recommended procedure is to keep the patient at bed rest and do daily spinal fluid taps so as to decrease the spinal fluid pressure.

CASE REPORTS

Mr. G. M. T., a 44-year-old white man, gave a long-standing history of intractable headaches. Nausea frequently accompanied the onset which usually occurred during the night. Postural change, particularly lying down, seemed to precipitate an attack or to aggravate one already established. Medication for pain was of little or no avail, the

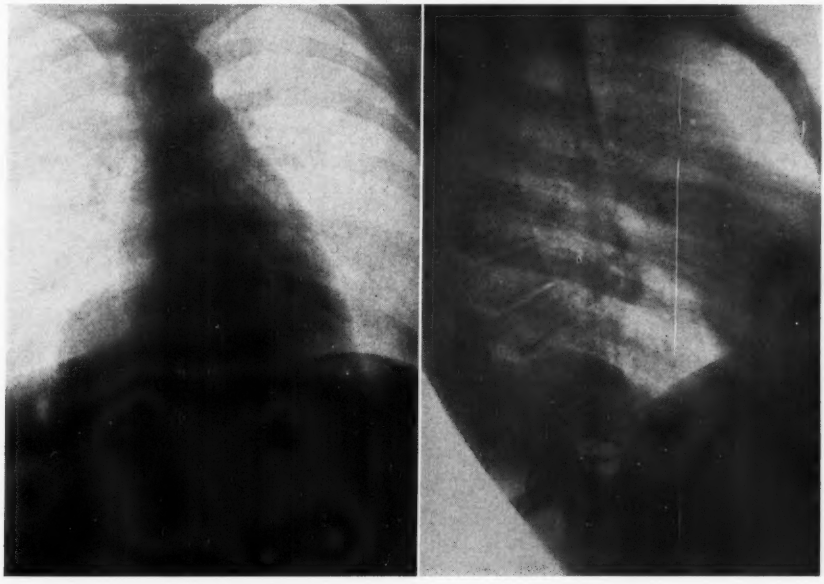

Fig. 3. Fig. 4.

Fig. 3.—Anteroposterior roentgenogram of the chest showing opacity in the right lower hemithorax in Case 2.

Fig. 4.—Lateral view of the right hemithorax in Case 2 showing meningocele.

greatest amount of relief being obtained by getting up and about. In fact, a headache would frequently subside after a day's activity. During the past 3 years, the patient had noted intermittent twitching of the eyelids and the right side of the face.

Physical Examination.—The skin of the thorax was the site of multiple cutaneous fibromata and areas of pigmentation. Intermittent twitching of the eyelids and musculature of the right side of the face was noted.

Roentgenographic Examination.—Posteroanterior and lateral chest films revealed a large tumor in the posterior mediastinum projecting to the right. The mass measured about 11 cm. in diameter, was smooth in contour, and extended from the sixth to the tenth vertebra. There was considerable decalcification and spreading of the proximal portion of the seventh and eighth ribs on the right. There was slight kyphoscoliosis with convexity to the left.

Diagnosis.—Neurogenic tumor of the posterior mediastinum.

Operation.—Exploration of the right pleural cavity revealed a large cystic mass occupying the mid-portion of the posterior mediastinum. The overlying pleura was dissected away from the cyst so as to gain adequate exposure. Dissection was carried down to an enlarged intervertebral foramen between T_5 and T_6. The cyst was then opened, spinal fluid evacuated, and the spinal cord amply visualized. The cyst wall, consisting of dura, was then trimmed, taking care not to sacrifice too much tissue. The enlarged intervertebral foramen was closed by imbricating the dural flaps, using 3-0 interrupted silk sutures. The dural repair was reinforced by imbricating the mobilized pleura in the same manner. Two No. 34 mushroom intercostal catheters were left in place for application of continuous suction during the immediate postoperative period.

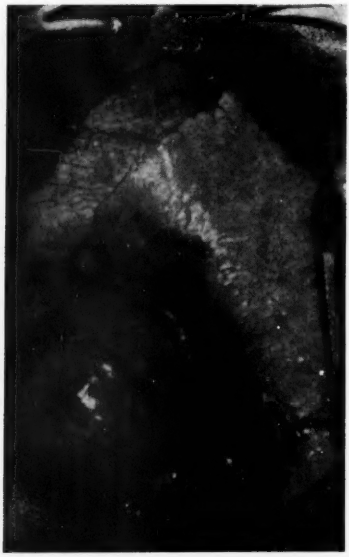

Fig. 5.—Photograph showing open hemithorax and meningocele prior to surgical repair.

Postoperative Course and Follow-up.—The patient's convalescence was uneventful. Eighteen months following surgery he reports definite diminution of the severity and frequency of headaches.

Mrs. B. A., a 37-year-old white woman, was admitted to the hospital for diagnosis and treatment. An opacity had been disclosed in the right lower hemithorax some months before as a result of a routine chest survey. She had noted some weakness in the right leg for several years. This had resulted in a slight limp. However, she had experienced this for so long a period of time that she paid little or no attention to it.

Physical Examination.—Slight weakness of the right leg was noted. Knee jerks were, plus 3 on the right, plus 1 on the left. Ankle jerk was absent on the right, present on the left.

Roentgenographic Examination.—Chest film revealed a round density measuring 6 cm. in diameter located just to the right of the vertebral column. There was some destruction of the bodies of T_{10} and T_{11}.

Diagnosis.—Neurogenic tumor, dumbbell type, arising in the posterior mediastinum.

Operation.—Exploration of the right pleural cavity revealed a large, rounded cystic mass communicating with a defect between the tenth and eleventh ribs. A small bridge of bone was present beyond which another sacular mass protruded. The meningocele was dissected free down to the neck of the sac which measured approximately 1.5 cm. in diameter. Upon opening the first meningocele, the second collapsed much as a pantaloon inguinal hernia would do. The edges of the resected sac were carefully imbricated with interrupted silk sutures. The larger meningocele was then excised at the neck and closed with two rows of 3-0 silk sutures, one row of mattress and the other row interrupted on the free edge. Two mushroom catheters were placed in the right hemithorax. These were connected to underwater suction drainage.

Postoperative Course and Follow-up.—The postoperative course was entirely uneventful. There is no evidence of recurrence of the meningocele over 1½ years after surgery, and the weakness noted preoperatively in the right lower extremity has not progressed.

Fig. 6.—Photograph showing opened meningocele in Case 2 in preparation for surgical closure.

SUMMARY

Intrathoracic meningocele is an anomaly usually associated with neurofibromatosis and typical skeletal changes affecting the thoracic vertebrae. The condition may be suspected or even anticipated whenever a posterior mediastinal tumor occurs in conjunction with skeletal changes and neurofibromatosis. The treatment is surgical, since it represents an expanding lesion. Reference is made to Table I and Table II for a summary of the cases encountered in the literature to date.

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Discussion of Two Meeting Papers

CREATION OF A TEMPORARY ARTIFICIAL DUCTUS FOR THE SURGICAL CORRECTION OF VENTRICULAR SEPTAL DEFECTS ASSOCIATED WITH SEVERE PULMONARY HYPERTENSION BY HOWARD D. SIRAK, M.D., AND DON M. HOSIER, M.D., COLUMBUS, OHIO,* AND CORRECTIVE SURGERY IN THE PRESENCE OF FAR-ADVANCED PULMONARY HYPERTENSION BY C. WALTON LILLEHEI, M.D., DONALD J. FERGUSON, M.D., AND RICHARD L. VARCO, M.D., MINNEAPOLIS, MINN.†

DR. WILLIAM H. MULLER, Jr., Charlottesville, Va.—Both of these are important papers because, as they indicated, pulmonary hypertension is one of the major problems that one faces in corrective surgery for congenital heart disease. About 5 years ago, Dr. Kattus and I had planned to use a decompressing shunt from the aorta to the pulmonary artery when we were closing a large ductus with reversal of blood flow. Fortunately, we were able to close it without performing a shunt. I am delighted to see that Dr. Sirak has utilized it with a high degree of benefit in his patients.

I would like to mention one other approach in the management of pulmonary hypertension in patients with congenital heart disease, that is, a procedure to narrow the main pulmonary artery in certain patients who have a physiologic single ventricle and severe pulmonary hypertension. This procedure was initiated before total cardiopulmonary bypass and open-heart surgery was being done. The indications for it at that time, in our opinion, were rather broad. We advocated its use to prevent the development and progression of luminal narrowing resulting from medial hypertrophy and intimal fibrosis of the small muscular pulmonary arteries and arterioles of the lung, as was shown in both these patients, and to control high output failure in small infants. We now think that its chief indication is in the small infant with a ventricular septal defect or a single ventricle where the mortality rate of a corrective open-heart procedure in our hands, and I believe in others, is excessive. At the present time, it is the only treatment I know of for a single ventricle, although correction of this defect has been tried unsuccessfully with total bypass. It may also be used in the older child who has passed from a balanced phase to an early right-to-left shunt, where the mortality rate from a corrective procedure may also be high.

Work in our research laboratory by Dr. Richard H. Blank appears to indicate that severe pulmonary vascular changes, which we have hitherto thought were permanent, are reversible. It is possible, therefore, that after a period of time patients who have had narrowing of the pulmonary artery may be able to have a corrective open-heart operation with a lower mortality rate.

(slide) This slide demonstrates a very large pulmonary artery and a small aorta, as we usually find it in these patients. (slide) Here, the main pulmonary artery is narrowed by suturing a band of synthetic fabric about it. We initially removed a segment of the circumference of the artery but now rely totally on the band.

One of the critical points of this operation is the degree of narrowing, and we find that the lumen of the artery must be reduced to one third of its previous size in order to obtain the necessary reduction in pulmonary blood flow. This may best be determined by measuring the pressure proximally and distally to the point of stenosis. While a

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†Not yet published.

normal pulmonary artery pressure is not often tolerated by the heart in most instances, it can be reduced from 30 to 40 mm. Hg. If this degree of pressure reduction is not obtained, the desired clinical improvement in the patient does not usually follow.

DR. CHARLES P. BAILEY, Philadelphia, Pa.—My discussion concerns not only these two papers but also, perhaps in anticipation, Dr. Kirklin's paper on the tetralogy of Fallot. We have been troubled not only by the operative mortality in cases of ventricular septal defect with pulmonary hypertension but also by the surprisingly high operative mortality which attends correction when there is no pulmonary hypertension. You have just heard Dr. Sirak's suggestion for the elective creation of a patent ductus arteriosus and acceptance of a two-stage procedure because he is having similar difficulties. Dr. Effler has described acute left atrial hypertension after definitive closure of defect and restoration of the heartbeat. It occurred to us that this might be related to the suddenness with which we altered the cardiac physiology. It seemed to us that it would be better if the defect, and hence the shunt, were closed off gradually.

(slide) Following the line of thought presented last year by Dr. John Lewis, we created a prosthesis made up of two attached slices of compressed Ivalon sponge, one of which is perforated centrally. This we call a prosthetic artificial foraminal valve. At first we thought of it only to permit continuation of the right-to-left shunt in a case of the tetralogy of Fallot completely corrected. This would tend to prevent engorgement of a hypoplastic pulmonary vascular bed.

We have applied this prosthetic valve in atrial septal defects, as you see from this case, with a large common atrioventricular canal. The defect measured 1½ in. by 1¼ in.

Some time ago we had the experience of observing a rise in the left atrial pressure to 80 mm. Hg, with ensuing death, in a patient with an uncomplicated ventricular septal defect immediately after closure and restoration of circulatory integrity. Effler and Dodrill have had similar experiences in which the left atrial engorgement was followed by uncontrollable acute hemorrhagic pulmonary edema. It occurred to us that the artificial foraminal valve could also be applied in such a manner as to permit a continuation of a pre-existing left-to-right shunt.

These prosthetic valves placed in an experimental animal are closed by sealing together of the leaves within 8 weeks. We think that Dr. Lillehei's suggestion as to a perforated Ivalon disc is particularly pertinent in cases of pulmonary hypertension with a nearly "balanced" shunt. However, in patients with the tetralogy of Fallot and in those with uncomplicated ventricular septal defect with unidirectional shunt, we feel that it is preferable to use a unidirectional valve of this type to provide immediate maintenance and gradual decrease of the one-way shunt until its final obliteration. After all, does it matter whether we cure the patient in 5 minutes or in 8 weeks? So far we have had one death in the application of this technique in 8 patients.

DR. OSLER ABBOTT, Emory University, Ga.—This discussion was not returned.—EDITOR.

DR. FRANK L. GERBODE, San Francisco, Calif.—I would like to discuss Dr. Lillehei's paper and to compliment him again on pointing the way in this particularly difficult field of pulmonary hypertension. I would like to ask whether he believes that the type of pulmonary hypertension found in patent ductus arteriosus, in which there are structural changes to be seen in the lung, is perhaps exactly the same as is found in some types of ventricular septal defect. The reason I ask this question is that we have found in a series of patients with pulmonary hypertension and associated ductus, that closure of the ductus does not necessarily lead to an eventual cure of the hypertension. We have at least 3 patients who are in serious difficulty with pulmonary valve insufficiency, with very large hearts, and we have had 2 patients who have died 6 months or later from complications of pulmonary hypertension.

The second point I would like to discuss is the use of the median sternotomy. I am very glad that Dr. Lillehei is now using this incision, remembering that Dr. Wangensteen advocated it many years ago. We have used it for about a year now, in approximately 87 open-heart cases, and have found that it is extremely useful and advantageous, particularly in patients who have pulmonary difficulties such as he has described. We have not been able to retain the integrity of both pleural spaces in many instances, and always drain the left pleural space as well as the right if it is entered. Another drain is always placed near the well-fenestrated pericardium to prevent tamponade.

It is said that one cannot expose a persistent left superior vena cava through this incision. This is untrue, for it is quite easy to bring it into view by retracting the left atrial appendage. A large ductus may be difficult to manage if there is gross cardiac and pulmonary artery enlargement, but an average ductus may be dissected intrapericardially.

DR. SIRAK (Closing).—This afternoon you have seen some suggestions for managing patients who already have severe pulmonary hypertension. Dr. Muller's discussion brings up the other side of the problem—that is, trying to interrupt this process before it reaches such an advanced stage. This problem could best be attacked during infancy. These very small patients already have varying degrees of vascular changes, but still have large flows. Unfortunately, the risk of open-heart surgery in this group is still considerable. Therefore, it would seem logical to approach this problem using a two-stage method.

I should like to show an example illustrating this approach. This is a child who, at the age of 2 months, developed heart failure and had recurrent pneumonia. A pulmonary "tuck" was performed at that time (1955). He did very well until 26 months of age when he began getting cyanotic and had a marked decrease in his activity tolerance. Although he had marked clubbing and cyanosis, he had gained in weight from 7 pounds (at the time of the first-stage "tuck") to 32 pounds at the time of the second-stage open-heart procedure. (slide) This is the appearance of the anatomy at the time of the open-heart operation for repair of the "tuck" and closure of the septal defect. (slide) Not shown here is the enormous amount of scar tissue which was present in the bed of the "tuck" area. This precluded dissecting out the pulmonary artery. I might add that there were very few adhesions in the pericardium. After closing the interventricular septal defect, a probe was passed via the right ventricular incision up through the constricted area of the pulmonary artery. The artery was then incised from the valve to its bifurcation, demonstrating a very narrow lumen (3 mm.) through the "tuck" area. A piece of aortic homograft was then sewn to the edges of the opened stenotic area to form a new roof, thereby restoring full patency to the pulmonary artery (slide). I think that the aortic homograft probably will do very well in such a low pressure area and that the diameter is adequate to carry him through adulthood.

(slide) This is the cardiac catheterization done just before the pulmonary "tuck." He had a moderate amount of pulmonary hypertension and the biopsy showed moderate vascular changes. You will note that the cardiac catheterization done just before the second operation for removal of the "tuck" and closure of the septal defect shows pronounced right ventricular hypertension with a gradient across the "tuck" area. The peripheral arterial saturation indicates how markedly cyanotic he had become. The last catheterization, performed 2 months after removal of the "tuck" and closure of the defect, shows obliteration of the shunt and normal pressure and saturation values in the right ventricle and pulmonary artery. (slide) This is a picture of the child 4 months after the definitive procedure.

I should like to ask Dr. Lillehei what kind of respirator he uses for complete control of respiration. Also, what is the size of the perforations in the Ivalon patch? He mentioned that he is now using the median sternotomy exclusively. We have also found this to be a very suitable exposure for all congenital lesions except those associated with a patent ductus.

The question is raised whether, in employing a first-stage aorticopulmonary shunt as we have advocated, the lungs might be further damaged by the persistence of the

left-to-right shunt. I do not believe that this is a problem because flow is reduced through the tubular graft and, furthermore, it is left in place for only a few months. Dr. Lillehei has advocated, in his paper, creating a shunt from the pulmonary artery to the left atrium in order to divert pressure away from the pulmonary arterial bed. By doing this, a tremendous flow-gradient would be established, thus inducing a large bleed-off from the pulmonary artery. I should like to ask Dr. Lillehei whether this might not seriously compromise pulmonary perfusion. The work of the left side of the heart would also be increased.

I would like to thank the discussers, my sponsor, and the Association for the privilege of the floor.

DR. C. WALTON LILLEHEI, Minneapolis, Minn.—This discussion was not returned.—EDITOR.